THE
LARYNGOSCOPE.

VOL. XLI APRIL, 1931. No. 4

PRACTICAL OTOLOGIC ANATOMY.*

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No attempt is made to describe the detailed anatomy of the temporal bone; that information can be gleaned from the many excellent textbooks of anatomy in the English and German languages. Rather, the purpose of this paper is to point out the practical application of a thorough knowledge of the temporal region in the daily routine of the otologist. To my mind there is no branch of surgery in which the results of any major operative procedure are so dependent on anatomic knowledge. Vital structures are crowded within a small space and the distance between disaster and success is measured in less than millimeters. It is hoped that the perusal of this article will not only be a short review but that it will stimulate the otologist to delve more deeply into this basic and fundamental subject of his specialty.

The anatomy of the temporal region is an absolute prerequisite for the intelligent treatment of diseases of the ear.

The component parts of the temporal bone are readily separated in the newborn and consist of the pars squamosa, pars petrosa and

*Illustrations drawn by the author.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication March 5, 1931.

the annulus tympanicus. These three parts are separated by well marked sutures, which gradually become obliterated, so that by the end of the third year the temporal bone practically assumes the adult configuration. The knowledge of their anatomic position is of great practical importance because these sutures are the routes for the passage of pus from the middle ear to the exterior, as well as to the middle cranial fossa.

The most important of these is the mastosquamosal externally and petrosquamosal on the inner or medial aspect at the junction of the squamous and petrous portions. The sutura mastosquamosa runs through the antrum tympanicum. This explains the frequency and

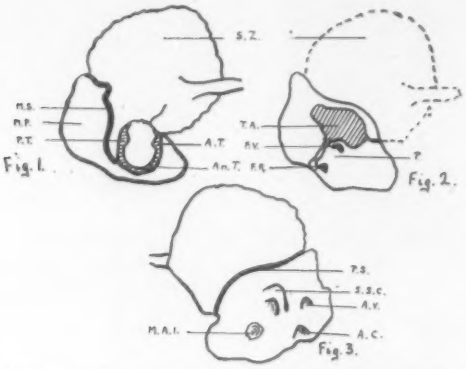

Fig. 1. Temporal bone at birth. M.S: Sutura mastosquamosa. M.P: Mastoid process. P.T: Posterior tubercle. A.T: Annulus tympanicus. An.T: Anterior tubercle. S.Z: Pars squamozygomatica.
Fig. 2. Temporal bone at birth. T.A: Tympanic antrum. F.V: Fenestra vestibuli. F.R: Fenestra rotundum. P: Promontory. The annulus tympanicus and the Pars squamozygomatica are detached.
Fig. 3. Inner aspect of the temporal bone. P.S: Sutura petrosquamosa. S.S.C: Superior semicircular canal. A.V: Aqueductus vestibuli. A.C: Aqueductus cochleae. M.A.I: Meatus auditorius externus.

rapidity of development of a postauricular subperiosteal abscess in children following a middle ear and antrum infection. The sutura petrosquamosa runs through the tegmen antri and tegmen tympani. In embryonic life it lodges a venous sinus which connects the sigmoid with the cavernous sinus. This sinus becomes obliterated after birth but the suture persists and in children a middle ear infection not infrequently gives rise to meningismus or mild meningeal symptoms because of the patulency of this suture. In the adult this suture is obliterated but in rare instances it persists and becomes a source of potential danger during a middle ear infection. Of practical importance also is the fact that the tegmen or roof of the tympanic

and mastoid antra is very thin and is the commonest site for a fracture of the base of the skull. Another suture is the petrotympanic (Glaserian) for the passage of the chorda tympani and blood vessels. Occasionally in the young child a middle ear infection will break through this fissure into the glenoid fossa, particularly in tuberculosis of the middle ear, and produce ankylosis and swelling of the mandibular articulation.

DIFFERENCES IN THE APPEARANCE OF THE TEMPORAL BONE IN THE ADULT AND AT BIRTH.

At birth the nipple-like shape of the mastoid is absent, there is no bony external auditory meatus and the facial nerve is exposed on the surface just back of the lower posterior border of the annulus tympanicus. In the middle ear the facial nerve lies in a groove

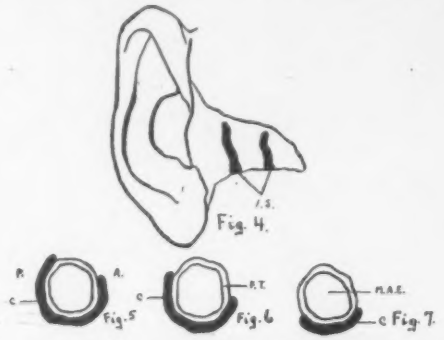

Fig. 4. External auditory meatus—cartilagenous portion. I.S: Incisurae Santorini.
Figs. 5, 6 and 7. Showing disposition of cartilage on the posterior inferior and anterior walls. P: Posterior wall. A: Anterior wall. C: Cartilage. F.T: Fibrous tissue. M.A.E: Meatus auditorius externus.

instead of in the facial canal, as in the adult. The sutures, as pointed out before, are well marked and patulous. It is also of practical importance to add that the tympanic membrane is placed almost horizontally instead of obliquely, as in the adult. This should be borne in mind in performing a myringotomy, because in sweeping the knife downwards the point must advance farther medially.

MEATUS AUDITORIUS EXTERNUS.

The external auditory canal is, roughly speaking, a little more than one-third of its length cartilaginomembranous and a little less than two-thirds osseous. The construction of the cartilaginous canal is interesting and of practical importance to the otologist. It must be remembered that the cartilage does not entirely surround the canal but that beginning at the cavum conchae it is incorporated in the

posterior, inferior and anterior walls and as it advances towards the bony meatus the cartilage disappears from the posterior and anterior walls, so that at its junction with the bony meatus, cartilage is only present on the inferior wall. In addition, this cartilage is broken up usually by two fissures which run transversely to the long axis of the canal—incisurae Santorini. It is because of this arrangement that the canal can be straightened out by pulling upwards and backwards in the adult and somewhat downwards and backwards in the child. This maneuver places the long axis of the entire canal in one straight line, permitting us to observe the drum membrane. Through the incisurae Santorini course blood vessels, lymph vessels and nerves,

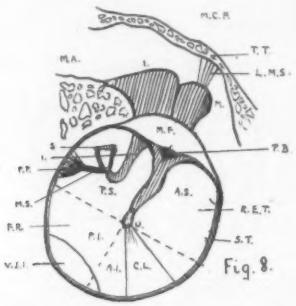

Fig. 8.

Fig. 8. Right tympanic membrane — the lateral wall of the epitympanum is dissected away, exposing the bodies of the malleus and incus. M.C.F: Middle cranial fossa. T.T: Tegmen tympani. M.A: Mastoid antrum. L.M.S: Ligamentum mallei superior. M: Malleus. I: Incus. M.F: Membrana flaccida. P.B: Processus brevis. S: Stapes. P.P: Prominentia pyramidalis. M.S: Musculus stapedius. F. R: Foramen rotundum. V.J.I: Vena jugularis interna. C.L: Cone of light. S.T: Sulcus tympanicus. R.E.T: Region of Eustachian tube. A.S: Anterior superior segment. A.I: Anterior inferior segment. P.S: Posterior superior segment. P.I: Posterior inferior segment.

but of importance to remember is that infection from the canal may pass through these fissures anteriorly into the region of the parotid and vice versa and posteriorly to the mastoid area. This helps to explain the not uncommon appearance of swelling of the parotid region and of swelling in the mastoid region in furunculosis of the cartilaginous canal, particularly in children. This arrangement also explains the exquisite pain on traction of the auricle in furunculosis of the canal and its absence in inflammations of the middle ear; in the infant, there being no bony canal, traction of the auricle produces pain also in middle ear diseases.

The bony canal is represented at birth by a ring, the annulus tympanicus, which shows a deficiency in its superior segment, the Rivinian notch—Incisura Tympanica. Two tubercles are present on its

external surface, the anterior and posterior tubercles. From these, aided by the squamous and mastoid portions of the temporal bone, the osseous canal develops.

In the adult the bony canal occupies a little less than two-thirds of the length of the whole canal. At the junction of the middle and inner thirds the lumen is narrowest—the isthmus. From here the lumen widens out again but the inferior wall rather sharply curves downwards. This knowledge is essential particularly in the treatment of foreign bodies. Most of the foreign bodies become lodged at the isthmus and this readily explains why syringing is the method of choice. Instrumental tampering may push the foreign body be-

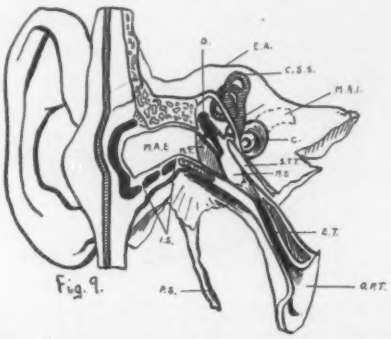

Fig. 9.

Fig. 9. Dissection showing the external, middle and internal ear. Semi-diagramatic view. E.A: Eminentia arcuata. C.S.S: Superior semicircular canal. M.A.I: Internal auditory meatus. O: Ossicles. C: Cochlea. M.A.E: External auditory meatus. M.T: Tympanic membrane. S.T.T: Semicanalis tensor tympani. M.E: Middle ear. I.S: Incisurae Santorini. E.T: Eustachian tube. O.P.T: Ostium pharyngeum tubae. P.S: Styloid process.

yond this narrowest part towards the tympanic membrane and thus make a fairly simple procedure one fraught with danger to the drum and contents of the middle ear.

THE TYMPANIC MEMBRANE.

Because of the fact that the anterior and inferior walls of the external auditory canal extend some 5 to 7 m.m. more medially than the posterior and superior walls the tympanic membrane is placed obliquely to the long axis of the canal. Actually, the matter is not so simple, but a good way to understand the position of the drum is to remember that the upper posterior part of the drum is nearer the line of vision of the observer than the anterior inferior. This point must be remembered in doing a myringotomy: when the incision is from above downwards (and to my mind preferable to making an

incision from below upwards), the point of the knife, perforce, must advance more medially as it sweeps towards the hypotympanum.

Anatomically and practically, the drum is divided into the pars flaccida, which is attached to the incisura tympanica—the Rivinian notch—and pars tensa, which is attached to the sulcus tympanicus. The drum is further divided by imaginary lines—a vertical running through the handle of the malleus and a horizontal running through the umbo. In this way four unequal segments are mapped out. This subdivision is of real practical importance because it helps us to visualize the location and position of the various parts of the middle ear. In addition, it furnishes us a clue as to the focus of suppuration of the middle ear in the presence of a perforation. Thus a perforation in the anterior portion points to involvement of the Eustachian tube. A perforation in the upper posterior segment points to involve-

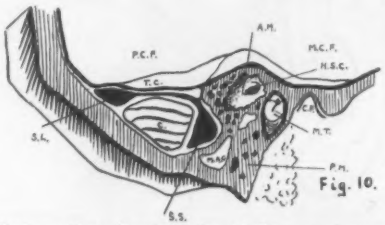

Fig. 10.

Fig. 10. Saggital section through the right ear, 4 m.m. lateral to the tympanic membrane. The relation to the middle and posterior cranial fossae, cerebellum and sigmoid sinus is clearly shown—from a dissection by the author. P.C.F: Posterior cranial fossa. M.C.F: Middle cranial fossa. T.C: Tentorium cerebelli. S.L: Sinus lateralis. C: Cerebellum. S.S: Sinus sigmoideus. A.M: Mastoid antrum. H.S.C: Horizontal semicircular canal. M.T: Tympanic membrane. C.P: Condyloid process. M.A.C: Mastoid air cell.

ment of the aditus ad antrum and mastoid antrum. A central perforation is indicative of suppuration of the mesotympanum and its contents.* A perforation in the pars flaccida points to suppuration in the attic or epitympanum with possible involvement of the ossicles, their coverings of folds of mucous membrane and the tegmen or roof of the middle ear. Perforations then in the anterior segment and around the umbo may be looked upon as relatively less dangerous than perforations in the posterior segment and Shrapnell's membrane.

The normal tympanic membrane presents a certain characteristic appearance. The processus brevis, together with the anterior and posterior folds, are very prominent. The deepest concavity is at the umbo, from which running anteroinferiorly is the cone of light. Ab-

*Anatomically the middle ear is divided into an epitympanum—the space above the prominence of the facial canal, a mesotympanum—the space between the prominence of the facial canal and lower border of the promontory, and an hypotympanum—the space between the lower border of the promontory and the floor.

sence of the pearly gray color, or of the cone of light, or of the characteristic appearance of one or more landmarks, denotes pathology. Hence the practical importance of being thoroughly familiar with the normal appearance of the tympanic membrane.

ORIENTATION OF STRUCTURES OF THE MIDDLE EAR.

For practical purposes the cavity of the middle ear may be looked upon as a room consisting of a floor, roof and four walls. Within this space are a number of structures easily identified. A very good way to form a mental image and permanently fix it in your mind is to imagine yourself standing in the right middle ear (as in a room) facing forwards. Then, in front of you is the tympanic opening of the Eustachian tube. Above it, on the medial wall, lies the tensor

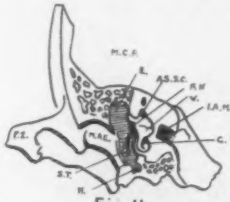

Fig. 11.

Fig. 12.

Figs. 11 and 12. Coronal section through the external canal and vestibule —from a dissection by the author. M.C.F: Middle cranial fossa. E: Epitympanum. A.S.S.C: Ampulla superior semicircular canal. F.N: Facial nerve. V: Vestibule. I.A M: Internal auditory meatus. C: Beginning of the Lamina spiralis ossea. H: Hypotympanum. S.T: Sulcus tympanicus. M.A.E: External auditory meatus. P. Z: Zygomatic process. S.S.C: Superior semicircular canal. A.E.A: Aditus et antrum. A.H.C: Ampulla horizontal canal. C.P: Common crus of superior and posterior canals. F.V: Foramen vestibuli. P: Promontory.

tympani muscle lodged in a bony semicanal. Just before the oval window its tendon turns at right angles, leaves the medial wall, traverses the middle ear and is inserted into the posterolateral surface of the neck of the malleus. Behind you is the hollow pyramidal process, from which the stapedius muscle emerges to go to the head of the stapes. Above this pyramidal process is the narrow passage—aditus ad antrum — leading to the mastoid antrum. The floor is curved and forms the roof for the jugular fossa in which the jugular bulb lodges. The roof or tegmen is a thin plate of bone and forms the floor of the middle cranial fossa on which the third temporal convolution rests. On your right is the tympanic membrane with the handle of the malleus incorporated within the drum. To your left is the promontory with the oval window above and the round window below. Above the oval window and running backwards is the promi-

nence of the facial canal and just above that is the hard, glistening, ivory-like prominence of the horizontal semicircular canal. The picture of the contents of the middle ear can now be completed by imagining that the space occupied by you is filled in with the ossicles.

In this connection it is well to remember that the head of the malleus and body of incus are in the attic or space above the tympanic membrane. And finally, this cavity, which in reality is some 12 to 15 m.m. in height, 10 to 15 m.m. in anteroposterior diameter, and 2 to 6 m.m. from side to side, is the most frequently involved in either acute or chronic diseases of the middle ear. One can therefore see the utmost necessity of being thoroughly familiar with the contents and configuration of the middle ear in the daily treatment of ear diseases.

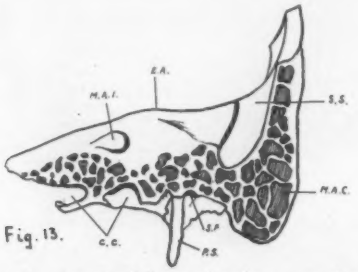

Fig. 13.

Fig. 13. Section of a temporal bone showing an unbroken chain of air cells extending from the mastoid process to the apex of the pyramid—In the author's collection. M.A.I: Meatus auditorius externus. E.A: Eminentia arcuata. S.S: Sigmoid sinus. M.A.C: Mastoid air cells. S.F: Stylomastoid foramen. P.S: Styloid process. C.C: Carotid canal.

THE EUSTACHIAN TUBE.

The Eustachian tube is the most frequent and the most direct route to carry infection to the middle ear. It runs from the anterior portion of the middle ear forwards, downwards and medially to its pharyngeal orifice on the lateral wall of the pharynx, about the level of the posterior tip of the inferior turbinate.

Physiologically, its main purpose is to ventilate the middle ear, as well as to equalize atmospheric pressure on both sides of the drum. In the adult it is about 36 m.m. (1½ inches) long, of which one-third is bony and two-thirds cartilaginous. Its tympanic orifice is half-a-centimeter in height and about 3 m.m. in breadth and is 25 c.m. (1 inch) above the pharyngeal opening. The lumen of the canal is of variable diameter—the osseous portion is 2 m.m., and at the junction with the cartilaginous portion it is even narrower. The cartilaginous portion widens out as the pharyngeal orifice is reached on the lateral wall of the pharynx.

The anatomy of this tube is different in the newborn. It is comparatively shorter and wider; its tympanic orifice is lower than in the adult, while the pharyngeal orifice is nearer the level of the soft palate. This places the Eustachian tube in the newborn in a nearly horizontal direction (against a downward direction in the adult). Hence, because of its peculiar anatomic position, particularly in the newborn and the young child, the Eustachian tube is the most frequent and the most direct route to carry infection to the middle ear.

And just as its anatomic structure enhances the development of

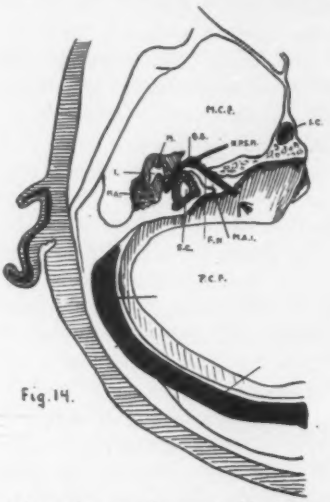

Fig. 14.

Fig. 14. Horizontal section of the pyramid. The roof of the internal auditory meatus has been removed and the facial nerve laid open. The tegmen tympani and tegmen antri are also removed, exposing the contents of the middle ear and the mastoid antrum — from a dissection by the author. M.C.F: Middle cranial fossa. P.C.F: Posterior cranial fossa. M: Head of malleus. I: Body of incus. M.A: Mastoid antrum. S.C: Semicircular canals. G.G: Geniculate ganglion. N.P.S.M: Nervus petrosus superficialis major. F. N: Facial nerve. M.A.I: Meatus auditorius externus. I.C: Internal carotid.

infective processes, this very position fortunately makes the Eustachian tube and middle ear easily accessible to the otologist, and by means of air pressure, fluids and volatile gases he attempts to restore the functions of the tube, the middle ear and in part the labyrinth.

THE LABYRINTH.

Admittedly the detailed anatomy of the labyrinth is complicated and the only way to master it is through numerous dissections and examinations of histologic slides. From the practical viewpoint, however, the matter is not so difficult.

We may look upon the petrous portion of the temporal bone as a triangular pyramid having a base, an anterior, posterior, and an inferior surface. The base forms the medial wall of the middle ear and mastoid antrum and has been already described. The posterior surface forms a part of the posterior cranial fossa. The structures in relation with it are the sigmoid and inferior petrosal sinuses—the superior petrosal sinus occupies the superior border of the pyramid—the posterior semicircular canal, the aqueducts of the cochlea and the vestibule, the internal auditory meatus and the cerebellum. The anterior surface forms a part of the middle cranial fossa. In relation with it is the inferior surface of the temporal lobe. In addition, the superior semicircular canal forms a prominent bony arch—eminentia arcuata. In relation with the apex of the pyramid are the semilunar ganglion, the sixth nerve, important in understanding the Gradenigo syndrome, and the caverous sinus. The other structures in relation with the apex, and they are many, play a secondary role in the work of the otologist. Within this pyramid and occupying but little more than the volume of a half-inch cube, lies the labyrinth.

The labyrinth is a system of membranous channels consisting of the semicircular canals, utricle and saccule, which serve as the peripheral end organs of orientation and equilibration, and the cochlea, which is the peripheral end organ of hearing. These channels are enclosed within a bony capsule, the outlines of which closely follow the configuration of the membranous labyrinth. To the otologist the location of the various structures are of utmost importance as they furnish a clue of the ordinary pathways of infection. Thus, infection of the middle ear usually reaches the labyrinth through the oval and round windows or through caries of the promontory, or the horizontal semicircular canal (the other and less frequent paths of infection are purposely omitted from this discussion). In turn, the labyrinth transmits infection through both aqueducts, the internal auditory meatus and the posterior saggital canal to the posterior fossa, while infection of the middle fossa is through the eminentia arcuata. While it is true that an infection may travel from the cranial fossae to the labyrinth through the same pathways as described above, the otologist usually is not called upon to treat these cases. The necessity then of mastering the anatomy of this region is of paramount importance to the progressive otologist.

THE SURGICAL APPROACH TO THE MASTOID ANTRUM.

After the periosteum has been retracted backwards and forwards the adult mastoid shows characteristic landmarks. These are the linea temporalis, which in a practical way gives the level of the floor of the middle cranial fossa, the posterior wall of the external audi-

tory meatus, the spina suprameatum—spine of Henley—and, posteriorly, the mastoid emissary vein may show.

The next surgical procedure is the approach to the mastoid antrum. Most textbooks describe imaginary triangles, which are the corridors or pathways to the antrum. To the beginner this geometric approach is confusing; to the practical man it is superfluous. There is one landmark which is constant and reliable. The spina suprameatum is in practically all cases very distinct—it may be poorly developed but it is always present. Chiseling or curetting posterior to it and following the external auditory meatus will bring the surgeon to the mastoid antrum.

ANATOMIC VARIATIONS OF THE MASTOID PROCESS.

In general, the mastoid outlines conform to a characteristic pattern but no two mastoids are ever alike. Thus the sigmoid sinus may almost touch the posterior wall of the external auditory meatus, the tegmen antri may be very low, the facial nerve may be superficial or run an abnormal course.

The disposition of the mastoid cells at times is such that it is virtually impossible to exenterate all. I have seen and dissected specimens in which pneumatic cells extended clear to the apex of the pyramid and around the labyrinth, cells which surrounded the facial nerve, the bony Eustachian canal and the internal carotid, and cells which extended way into the squamous, zygomatic and occipital bones. This, by the way, is an answer why the route of approach to a simple mastoid, and particularly to a radical mastoid, through the external auditory meatus recently advocated is anatomically at least not feasible and surgically productive of numerous postoperative failures. It is evident then that the more the otologist familiarizes himself with the average normal mastoid the better he is able to cope with the abnormalities which present themselves not so infrequently at the operating table.

SUMMARY.

A short description of the temporal region has been given in the preceding pages with a view to point out to the otologist the utter necessity of being familiar with the anatomy of this region in order to intelligently treat diseases of the ear. It is hoped that he will be sufficiently stimulated not only to read anatomy but that he will make a pilgrimage at least once a year to the dissecting room. It is a combination of reading and numerous dissections that will enable him to gain a thorough and comprehensive knowledge of the temporal region.

60 East 96th Street.

PRELIMINARY REPORT OF A METHOD OF STUDYING THE DEVELOPMENT OF THE EAR, THE PARANASAL SINUSES, AND THE VARIOUS CENTRES OF OSSIFICATION IN THE HEAD.*

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In the Department of Oto-Laryngology, Washington University Medical School, there is being established an embryologic library of human specimens and their detailed descriptions. The department now possesses 30 human embryos, ranging from a rare specimen of 2¼ m.m. to one of 230 m.m. The program comprises: *1.* Measuring from rump to vertex; *2.* photographing the head and neck in frontal and lateral positions with a millimeter rule showing behind the specimen; *3.* X-raying in three positions: frontal, lateral, verticomental; *4.* serial sectioning; *5.* describing in detail each section, as well as the X-ray impressions.

It is hoped that the library will prove of value in oto-laryngologic research.

Although one can study the osteogenesis of the various structures in the stained section, one has in the embryologic X-ray a picture of all the structures and their relations to each other, at the various stages of development. The transparent specimen is valuable, but does not lend itself so well to publication and its processing would interfere with subsequent sectioning.

In order to stimulate others to utilize this interesting method of investigation, a preliminary report is presented. Please use a magnifying glass in studying the illustrations.

DESCRIPTION OF TWO SPECIMENS.

1. Human Embryo, 18 weeks, 130 M.m. (Rump-Vertex).

Fig. 1. Anterior-posterior radiogram. Note that the mandibular division of the first arch has ossified except at the site of arch junction anteriorly. The nasal capsule, which originates in the sphenoid, shows bone formation except at one small point superiorly. The superior maxilla (upper portion of first branchial arch) shows ossi-

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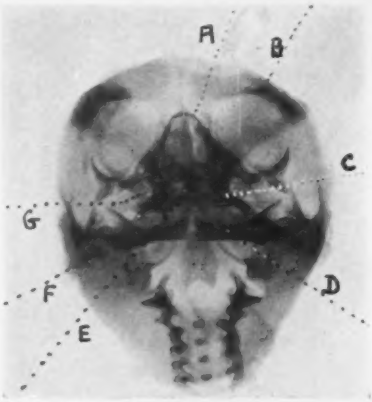
Editor's Note: This ms. received in Laryngoscope Office and accepted for publication Mar. 19, 1931.

fication in the region adjacent to the nasal cavities and to some extent laterally. There is no previous cartilage here. The maxillary sinus shows plainly but is quite small. The palatine processes of the



Specimen 1. Fig. 1. Anterior-posterior. A. nasal capsule, B. frontal ossification, C. maxillary sinus, D. mandible, E. palatine processes of superior maxilla, F. semicircular canal, G. superior maxilla.

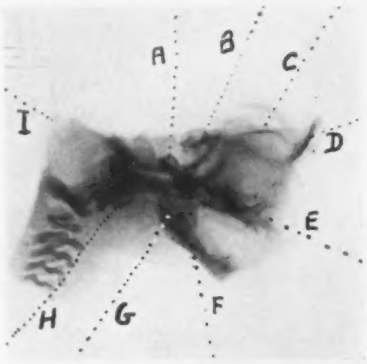


Fig. 2. Lateral. A. cochlea, B. sphenoid, C. nasal capsule, D. frontal, E. superior maxiliary, F. mandible, G. Meckel's cartilage, H. semicircular canal, I. occiput.

superior maxilla have fused and shows bone formation. Two areas of ossification are plainly developed in the frontal region. Semicircular canals show beginning ossification.

Fig. 2. Lateral view of same specimen. The frontal, superior maxillary and mandibular ossification is nicely seen. In the mandibular arch, Meckel's cartilage to form the malleus and incus, may be seen

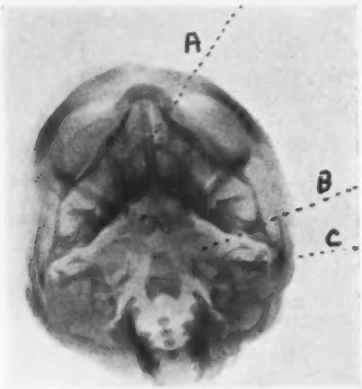
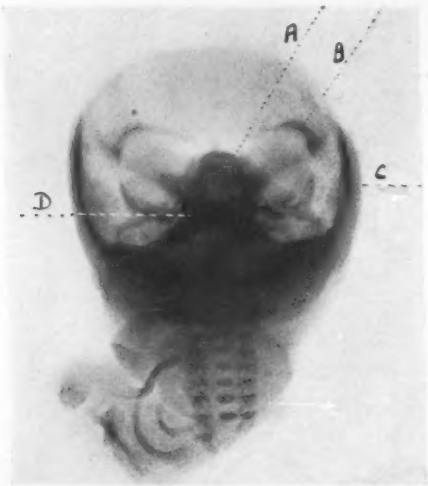


Fig. 3. Verticomental. A. vomer, B. cochlea, C. semicircular canal.



Specimen 2. Fig. 1. Anterior-posterior. A. nasal capsule, B. frontal, C. squama, D. antrum.

extending toward the ear region. The semicircular canals and cochlea show beginning of the bony periotic capsule (cochlea is further advanced than the canals). No ossification is present in the occipital

region. The sphenoid shows a narrow line of ossification with beginning sella formation. The nasal capsule is seen as a curved line.

Fig. 3. Verticomental view of same embryo. The vomer, with

Fig. 2. Lateral. A. occiput, B. semicircular canal, C. cochlea, D. frontal, E. nasal capsule, F. sella, G. sphenoid.

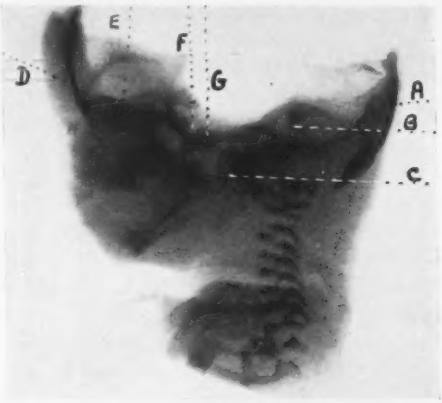


Fig. 3. Verticomental. A. frontal, B. vomer, C. parietal, D. tympanum, E. semicircular canal, F. occiput, G. cochlea.

beginning ossification on either side of its groove, is plainly visible. In this view we appreciate how little ossification has occurred about the cochleae and semicircular canals.

II. Human Embryo, Age 24 Weeks, 185 M.m. (Rump-Vertex).

Fig. 1. Anterior-posterior view: The nasal capsule shows little change over Specimen I. The superior maxilla shows a marked increase in bone formation and the antrum is much larger. Squama and parietal regions show well advanced ossification.

Fig. 2. Lateral view: The occiput show an advanced ossification, the frontal a marked increase over Specimen I. Sphenoid ossification is about three times as great as in No. I and the sella is now well developed. Here we can note the nasal capsule emerging from the sphenoid. A marked advance in ossification of the periotic capsule is plainly visible if we compare No. I and No. II.

Fig. 3. Verticomental view: With the ossification well along in the frontal, parietal and occipital regions the entire skull is now rimmed with bone.

Both labyrinths are visible and if we compare them with those in Specimen I we are immediately impressed with the very definite increase in density.

In conclusion, this very brief presentation is made simply as a suggestion to others working in this field. As our technique improves and more detailed study of a large series of radiograms is made, it is hoped that something of value may come from the investigation.

The radiograms are the work of Miss Rosaline O'Brien.

1000 Carleton Building.

FRACTURE OF THE SKULL COMPLICATED BY DRAINAGE OF SPINAL FLUID FROM THE EAR.*

DR. ALBERT B. MURPHY, Everett, Wash.

In the literature and in textbooks, comparatively little attention has been paid to involvement of the ear in fracture of the skull. There are a few reports of the remote effects and a few reports on mastoiditis complicating fracture of the skull. The complication of drainage of spinal fluid, a most interesting and at times provoking complication, has very few references in the literature, and practically no reference is made to this condition in the textbooks on otology. This report includes the study of cases seen with this complication.

In the cases of fractures involving the base of the skull observed since 1924, there has been drainage of serous exudate which was considered to be spinal fluid through the external auditory canal in thirteen instances, and in addition several cases have been seen some time after the injury in which the history would indicate that there had been drainage of spinal fluid. These cases have been studied as to cause, diagnosis, immediate and remote symptoms produced, mode of treatment and prognosis. In four of the cases in which the patients did not recover from the initial shock, and in those seen some time after the accident, the observations necessarily were limited.

In ten cases the point of trauma could definitely be localized. In eight of these the injury was in that quadrant of the skull occupied by the temporal and occipital bones on the side of the fracture: in the other two cases it was situated over the vertex. In two cases there were so many contusions that it was impossible to designate the causative one; in one case the patient was pushed backward to the floor and external evidence of trauma could not be found. The degree of trauma varied from a simple fall to the floor to a fall from a height of 40 feet, the patient striking the ground head first.

The diagnosis of drainage of spinal fluid from the ear is not easy to establish. When the middle ear has been traumatized there can

*Read before the Pacific Coast Oto-Ophthalmological Society, Victoria, B. C., Sept. 5, 1930, and before the Resident and Ex-Resident Physicians of the Mayo Clinic, Rochester, Minn., Oct. 8, 1930.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication Feb. 27, 1931.

be enough reaction to the lining membrane to produce considerable exudate. At first, the secretion may be stained with blood and will change at the end of 24 to 48 hours to a straw-colored or clear fluid, or it may be clear from the beginning. The amount of fluid secreted varies; in some cases there is fluid enough to saturate a wick in half an hour and other cases the canal will fill while under observation. In one case I was able to collect 20 c.c. of spinal fluid from the canal in ten minutes with a medicine dropper. In order that the spinal fluid drains from the ear, it is not necessary to have direct continuity by fracture from the middle fossa to the canal by tears in the dura and the drum membrane. Observation of a patient after operation for exenteration of the Gasserian ganglion for trifacial neuralgia taught me that the continuity does not even have to extend to the middle ear. In this case, in the course of exposing the floor of the middle fossa, some zygomatic mastoid cells were encountered and opened. The spinal fluid seeped down through these cells into the antrum and then to the middle ear. The patient complained of fullness in the ear immediately after the operation, and about the twelfth day after operation enormous amounts of spinal fluid began to drain from the ear. It is not known whether the pressure of the spinal fluid or the superimposed infection ruptured the drum membrane. Although an absolute diagnosis of drainage of spinal fluid cannot be made, I believe that the quantity of fluid secreted is the best criterion. As in rhinorrhea, one would think that the analysis of the fluid, particularly as to its sugar content, would be of great assistance, but this is not so, because the chemical constituents of other exudates from the ear has not been sufficiently established.

The Roentgenogram is a definite help in establishing the fact that a fracture is present, but of little help in determining its extent. Roentgenograms were made in 11 of the 12 cases; in one case the exposures were made after death. Fractures were observed in nine of the 11 cases, but in some cases the fracture was found only after repeated Roentgenograms had been made, as it was felt that fracture must have been present because there was drainage of spinal fluid. In one case five different Roentgenograms were made, but the fracture was seen in only one set. In the two cases in which we were unable to demonstrate the fracture, lateral Roentgenograms were taken. That was four years ago, at which time only one lateral view of the skull was taken. At present, I feel that if stereoscopic pictures of each temporal bone are taken without superimposition, the results will be even better. However, I doubt the wisdom of anyone making the statement that there is no fracture because it cannot be demonstrated by means of the Roentgen ray.

Extravasation of blood into the temporal muscle, independent of local trauma, with discoloration of the skin over this area, is of definite value in diagnosis. This was present in 75 per cent of the cases. Injuries to the nerve, such as those evidenced by total deafness, facial paralysis, aphasia and nystagmus, are of help in diagnosis, but it must be borne in mind that any of these can be caused by injury to the substance of the brain without fracture. The Barany test for sensitivity of the labyrinth and equilibratory division of the eighth nerve is very valuable, but should not be used too soon as an aid in the diagnosis, as there is the possibility of forcing infection inward.

Until the initial shock is overcome, the mode of treatment does not differ from that of skull fractures in general. Treatment of shock should include complete rest for the patient unless there are neurologic signs of a depressed fracture. The patient should not be left on a truck in the admitting room waiting for an ear examination or be moved about unnecessarily for Roentgenograms. At present, I paint the auricle and external canal as far as the isthmus or to the blood clot, if there is one present, with tincture of iodin, and apply a dressing to the mastoid. This dressing is changed once or twice in 24 hours, depending on the amount of drainage. I mention this as the procedure I use, but have no particular reason to believe that the incidence of infection will be less than with other procedures commonly used. In general, irrigation and swabbing are believed to be detrimental.

In two of the 13 cases there was evidence of superimposed infection by a change of the secretion to pus. I report one of these cases in detail, and give a brief resumé of the other:

Case 1: A man was pushed backward, and the occipital region struck the floor. He was comatose for 12 hours after admittance to hospital. The fluid from the left ear, which was blood-stained at first, became clear and very profuse. During the first five days the patient suffered considerably from headache and was unconscious for periods which bore direct relationship to the amount of spinal fluid draining. These attacks could be shortened and the drainage increased by elevation of the foot of the bed. On the sixth day the pain localized in the ear and the secretion became purulent, thick and foul. The temperature became elevated and there was definite blurring of the margins of the optic disks of both eyes, but in spite of the alarming local condition of the ear the patient's general condition improved in the next few days. Drainage from the ear continued for three weeks, running the course of acute otitis media

with mild mastoid involvement. From then on the recovery was uneventful.

Case 2: A boy, age 14 years, was struck by an automobile. There was a linear fracture extending downward through the mastoid process, from which clear spinal fluid drained through the middle ear for 12 days, after which it became purulent. At this time localized pain developed in the ear and over the mastoid, and the temperature became elevated. On the fourteenth day there was definite swelling over the mastoid process and the posterior superior canal wall was edematous. On the same day projectile vomiting began, and continued with all of the other symptoms until the sixteenth day, when the patient underwent an operation. At operation a perforation of the cortex of the mastoid was found over the postmeatal triangle. This lead was followed down to the antrum, the aditus was enlarged and all mastoid cells were removed. The incision was continued upward, exposing the fracture, which extended into the canal. The bone in this area was healthy and was not disturbed. A petrolatum pack was inserted in the antrum and the wound was closed. The ear became dry the third day after operation, when the posterior drain was removed and the wound was allowed to close. Recovery from this point was uneventful.

The average duration of drainage of spinal fluid was seven days in the cases that were not complicated by infection. The longest was 18 days, and this finally was controlled by elevating the head of the bed. During the period that increased intracranial pressure is a factor, the drainage of spinal fluid acts as a decompression and there is no reason for attempting to prevent its flow; on the contrary, it may be advisable to encourage it by elevating the foot of the bed.

The hearing of eight of the nine patients who recovered has been tested, and of several additional patients who were not seen at the time of the injury. In two cases there was total deafness; in two other cases both of the upper and lower tone areas were slightly affected, and in the remainder the hearing was affected in the upper tone areas. The type of deafness predominating usually resembles that of senile or boilermaker's deafness. That is, there is diminution of the upper tones without diminution in bone conduction.

There was no response to the Baranv test in one of the two cases in which there was total deafness, and this has been permanent. In the other case, temporary facial paralysis occurred. Two of the patients still complained of vertigo two and three years after the injury. This symptom was noted particularly on changing the posi-

tion of the head quickly in the dark and could be demonstrated clinically by dropping the head backward with the eyes closed. Three of the patients still complained of tinnitus several days after the injury, and I believe this will be permanent. One patient with definite aphasia has completely recovered.

It is difficult to determine if the disability will be permanent in these cases. In all but one case the earning capacity is practically the same as before the injury, but many of the patients have periodic attacks that disable them from one to two weeks. These attacks are characterized by headache, vertigo and tinnitus. The treatment of choice for these remote attacks consists of complete rest in bed, restriction of fluid intake, and phenobarbital administered in doses of 1½ gr.

CONCLUSIONS.

A serous discharge from the ear following trauma is usually, but not always, to be considered as drainage of spinal fluid.

Failure of the Roentgenograms to show fracture of the temporal bone is not conclusive evidence that there is no fracture present.

Acute mastoiditis may complicate fracture of the temporal bone.

Following a fracture of the skull, involving the temporal bone, there can be true vertigo with normal Barany reactions.

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VINCENT'S INFECTION OF THE MIDDLE EAR AND MASTOID; RADICAL OPERATION.*

Dr. R. A. Luongo, Philadelphia.

The following case seems worthy of record since Vincent's infection of the ear and mastoid is rare and the cases recorded are few in number: Mrs. L. F., age 23 years, in the fall of 1928 complained of discharging right ear since childhood and right frontal and temporal headaches of three months' duration.

On examination I did not find any local tenderness over the frontal, temporal or mastoid regions. The right cervical glands were enlarged. A profuse foul, bloody discharge came from the right ear. The external auditory canal was swollen and tender, and it bled at the slightest manipulation. The drum was almost completely destroyed: there was only part of the Shrapnell's membrane left. Smears taken from the right ear showed numerous Vincent's organisms (spirillum and B. fusiform). Several smears taken from the nose and throat were always negative for Vincent's organisms.

Treated with local applications and endovenous injections of neoarsphenamin, the discharge diminished, but the headaches became worse. About three weeks later the patient complained of pain in the right mastoid region, where tenderness was promptly elicited. The X-ray showed an extensive sclerosis of the right mastoid; there was no evidence of cells.

Vincent's organisms have the tendency to cause extensive bone necrosis. Dr. Yates has reported a case of Vincent's mastoiditis, where he performed a mastoidectomy and found the dural plate, the sinus plate, the posterior bony canal wall and the ossicles all well necrosed[1]. There are two cases reported in which fatal meningitis followed a Vincent's mastoiditis[2, 3].

Therefore, in my case, considering that the destructive Vincent organisms were undermining a solid sclerotic mastoid, for safety's sake I performed a radical mastoidectomy. At the operation I found hard, solid, sclerotic bone through the whole thickness of the mastoid; necrosis, fetid pus and granulations in the neighborhood of the aditus ad antrum; the posterior bony canal wall and the ossicles partly necrosed.

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*Read before the Philadelphia Laryngological Society, Feb. 3, 1931.

Editor's Note: This ms. received in Laryngoscope Office and accepted for for publication Feb. 15, 1931.

The postoperative treatment consisted of routine plus local applications of a 10 per cent solution of neoarsphenamin by means of saturated strips of gauze left in the canal and applied externally onto the wound.

Smears, taken every other day from the canal and the wound, continued to be positive for Vincent's organisms. In spite of this the patient made a splendid recovery and she was discharged from the hospital in two weeks, with the headaches cured and the aural discharge reduced to a minimum.

The treatment was continued, at intervals, with neosalvarsan for more than two years, and the smears, which were taken right along during this period, were always strongly positive for Vincent's organisms.

Fig. 1. Microphotograph of the smear taken lately from the ear. Spirochetae and B. fusiforms seen.

Others have had very encouraging results: R. Motta has reported two cases cured in a short time with only local applications of neoarsphenamin[4]. L. H. Barenberg and J. M. Lewis have reported three cases cured in 48 hours with local and systemic treatments of sulpharsphenamin[5].

The mastoidectomy and the specific treatments have greatly contributed to make my patient comfortable for more than two years and to reduce the aural discharge to a minimum quantity, but for the last three months she has been complaining again of the frontal and temporal headaches. Two weeks ago I started her on very intense specific treatment and it has helped the headaches and discharge considerably.

A study of the cases recorded reveals the following facts:

1. All cases occurred in children between the ages of 2 and 4 years, one of 12; mine is an adult.

2. The infection in my case is of more than two years' duration. When did it begin? How long will it last? How will it end? Yates says that Vincent of the ear is slow, insidious, almost symptomless, but very destructive[1].

3. The ears which were cured of Vincent's infection still remained chronic discharging.

4. All cases have histories of chronic ears, except one where the Vincent infection complicated an acute ear, which was perfectly cured with the specific treatments alone.

5. All cases, at the onset of the Vincent infection, showed a marked increase in the discharge, which became very foul and tinged with blood.

6. All had swollen, tender external auditory canals, which bled at slight manipulation.

7. Two cases had remote histories of Vincent's angina, one had an active Vincent's infection in the throat, while a similar infection was active in the ear. The other cases had neither any active infection of the nose and throat nor any previous history of any Vincent's angina.

8. All had enlargement of the cervical glands.

9. Two of them had ulceration of the canals, one of the auricle.

SUMMARY.

Vincent's infection of the ear is rare. Very few cases are recorded and in the textbooks little or no mention at all is made of this disease.

Etiology: The organisms are the same that are found in the throat. They will not grow on any of the usual media, so the slide must be examined, and use strong Ziehl-Nielson carbol-fuchsin: stain deeply to bring out the spirochetae.

The infection may enter the middle ear through the Eustachian tubes or, by contamination, through the external auditory canal. It usually occurs in children between 2 and 12 years of age. It is not a primary condition; it is usually secondary to chronic suppurative otitis media.

Symptoms: There may not be any symptoms until there is quite an involvement. Swelling of the cervical glands is an early symptom. Swelling of the auricle and external auditory canal may be present. The chronic discharge increases markedly and gets very foul and

tinged with blood. The canal is painful and bleeds at the slightest manipulation. One or more ulcers may be seen on the auricle and in the canal. The ulcer has the same characteristic of the one seen in the throat: it is covered with a dirty grayish pseudo-membrane and, when this is removed, one sees a sharply defined ulcer, which bleeds easily. There is no fever, no systemic symptoms. It has to be differentiated from T. B., diphtheria and foreign bodies.

Complications: Mastoiditis, meningitis and brain abscess.

Prognosis is good if treated early.

Treatment: Neoarsphenamin or sulpharsphenamin locally, 5-10 drops of a 10 per cent solution into the ear every 3-4 hours, and intramuscularly or endovenously, 0,3 to 0,6.

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1713 Pine Street.

TESTING FOR PERCEPTIVE DEAFNESS.*

DR. B. M. BECKER, Brooklyn.

In order to test the auditory function of each ear separately by air conduction we resort to one method or another in order to prevent any sounds which we employ at the time for testing purposes from reaching the cochlear portion of the ear opposite the one under examination.

Two methods are in common use to accomplish this result, namely, a wet finger introduced snugly into the ear canal, or the Barany noise apparatus. The one depends for its efficacy upon occlusion of the external ear, thereby preventing sounds from reaching the tympanum and carried to the inner ear; the other depends both on occlusion and noise for its results; the latter of which so overwhelms the conductive and perceptive parts of the ear, drowning out almost all extraneous sounds, that for all practical purposes it may be regarded as soundproof. The sounds perceived with the use of the noise apparatus in each ear are: the screaming voice at the ear, Galton's whistle and all the fork sounds, beginning with 512 and going up the scale.

How are we, with a similar degree of accuracy, to obtain in bone conduction tests results which we get in air conduction tests by ruling out one ear completely while examining the other?

It is quite obvious that with our standard tests, such as Weber, Schwabach and Rinné, we can arrive at a fair degree of certainty, but it falls short of the measure of reliability we obtain for each ear in air conduction tests. This would be possible only in the event of our being able to eliminate the perception of sound in one labyrinth while testing the other. For this reason, it seems to me, the standard tests furnish inconclusive data upon which to base a positive diagnosis of unmixed perceptive deafness, especially complete unilateral perceptive deafness.

If a patient should present himself complaining of impairment of hearing or complete deafness in one ear, the Weber test would furnish us with reliable data only when lateralization of the fork sound is to the affected ear. Then we may rightly assume that he suffers from a conductive or mixed lesion. On the other hand, when lateralization is to the opposite side we are not warranted in excluding a

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Editor's Note: This ms. received in Laryngoscope Office and accepted for publication Feb. 27, 1931.

conductive lesion nor are we warranted in pronouncing it a nerve lesion, for in this event we may be confronted with a mixed lesion in which the conductive or perceptive element may predominate.

We may with good reason draw a parellel between the auditory acuity of the cochleae and the results obtained by Stenger's two-fork test. When the sound is localized in the ear nearer the fork we do not maintain that the other ear is deaf because of its inability to perceive the fork sound. In the same manner, if one ear be slightly affected by a conductive lesion and the other normal and two vibrating forks of the same rate of vibration be held equidistant from both, within their radius of hearing the sound would be heard better or lateralized in the normal ear, depending upon the amount of obstructive deafness in the other ear. For these reasons the Weber would be lateralized to the sound ear should there be a mild affection of the cochlea on the opposite side, giving a reverse result from that obtained in purely conductive lesions. Even in normal bilateral hearing some will refer the Weber to one side or the other, due probably to a lack of sense of direction or to anatomical differences in the two sides of the skull, whereby one side will be more favorable for sound conduction or perception.

The Schwabach test suffers from similar defects. When a vibrating tuning fork is set against the mastoid antrum of an ear, the function of which is completely destroyed, the patient will still hear the sound of the fork. Of course, it is heard by the opposite ear, but the patient cannot always distinguish or point definitely to where he hears it. The tendency with many people is to refer it to the region where the fork makes contact. The same applies to the Rinné test.

If a means could be discovered to rule out one labyrinth while testing the other, as we do in air conduction tests with the Barany instrument, we could arrive at a greater degree of certainty as a result of our tests.

In considering the two methods of sound elimination by the finger and Barany's apparatus, I pointed out that the latter overwhelms the perceptive apparatus with its noise; that is, depresses its function to some extent. Therefore, I attempted to utilize this for sound elimination from one labyrinth while testing the other, but with disappointing results. For the sound of the 128 fork when forcibly struck is distinctly heard at either mastoid antrum while using a noise apparatus in each ear.

While thus considering a mode of sound elimination I chanced upon this simple device. When a stream of air is directed steadily

and forcefully against the tympanic membrane all sounds by bone conduction are eliminated and all the ordinary sounds used in air conduction tests, with the exception of the screaming voice at the ear and the upper limits of the Galton whistle.

The method is used in the following manner: A soft rubber tube of any convenient size, one end of which contains an ear tip, is placed very loosely in the ear canal opposite the one to be tested. The other end, either free or in which a small piece of glass tubing is inserted, is taken between the lips of the examiner, patient or assistant and a steady, forceful current of air is directed against ths drum while a forcefully vibrating fork is placed over the mastoid antrum of the ear under examination.

One should make certain of a free return flow of air from the canal.

If the examiner cannot communicate with the patient during this process he should explain to the patient beforehand what he is to take note of and express it by raising a finger, or in any other manner agree upon before, or he may reserve his answers to the finish.

If the patient hears the fork sound according to the method described there cannot be a shadow of a doubt that he heard it on the side under test; if he fails to hear it we are reasonably certain that we deal with complete loss of function or a grave functional condition of the cochlear portion of the labyrinth tested.

4802 18th Avenue.

PERIBULBAR ABSCESS—TWO CONSECUTIVE CASES.*

Dr. Louis R. Effler, Toledo.

During the last ten years, the writer has failed to encounter any cases of peribulbar abscess in the course of routine practice. During this time a fair number of mastoids have been operated. Recently there have been encountered two consecutive cases of peribulbar abscess. The fact is of sufficient importance to warrant this report. Fortunately, both cases recovered without incident. The starting feature in both cases was the fact that the indication for operation was subperiosteal abscess. The second feature was the fact that smears of pus in each case showed the streptococcus as the offending organism.

Case 1: A boy, age 6 years. Seen first on Oct. 19, 1930, with earache, right. The lad had contracted a severe "cold" one week before, probably influenzal. The right ear was paracentesed at first visit. For two weeks thereafter a profuse purulent discharge from this ear was noted. Quite early there was mastoid tenderness, especially over the tip and the antrum. The temperature during this time ranged between 101° and 104°. At the end of the second week, it was evident that the child was not progressing. The nurse on the case reported one chill, with several suggestions of vague chilly sensations. Discussion of the case with the parents resulted in the decision that, unless an unusual improvement was encountered within the next few days, the question of operation would have to be faced. In the meantime the writer suggested consultation. Consultation concurred in his viewpoint and decision. Strangely, within 24 hours after consultation a large swelling appeared over the body of the right mastoid—a subperiosteal abscess—that indicated operative interference without further delay.

At operation, on Nov. 6, the mastoid-labyrinth was found broken down in all directions. The involvement was unusual for so short a time. In addition to the breaking down in the cortex with discharge of pus externally there was involvement occipitally. A secondary incision at right angles to the original incision was found necessary to encompass a large area of osteomyelitis of the skull. Fortunately, this involved only the outer table, though it was entirely

*Cases presented before the Ear, Nose and Throat Section of the Toledo Academy of Medicine, Jan. 30, 1931.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication Feb. 11, 1931.

outside the area of the mastoid cells. The lateral sinus was found bathed in pus. It was followed down below the knee into a self-dissected abscess cavity filled with pus and granulations in immediate contact with the jugular bulb. This whole area was widely exposed, thoroughly exenterated and packed with iodoform gauze. A mild postoperative reaction took place. From the fifth day on, however, the child seemed out of danger. It was only a matter of time until he was completely recovered.

Case 2: A lad, age 16 years. Practically the same history was elicited of a heavy "cold," probably influenzal, of three weeks' duration. Almost at the start of the "cold" the right ear had perforated spontaneously and had discharged copiously for about two-and-one-half weeks. A rather sudden cessation of discharge at this point was followed in 48 hours by a large swelling over the right mastoid. This obvious subperiosteal abscess was diagnosed by his physician, who referred him to the writer on Jan. 15, 1931. There was no definite history of chills. Operation, of course, was promptly suggested. At operation, on Jan. 16, a most unusual anatomical anomaly of the cortex was noted. The temporal ridge was exceptionally prominent and rough. It was elevated fully half-an-inch. Its lower border projected or overhung the mastoid proper like a ledge, along which extended a deep groove. In this groove was a breaking down of the cortex, which discharged pus. The uncapping of the cortex revealed widespread destruction of the whole mastoid - labyrinth. Again the destruction extended far into the occipital area and also high under the prominent temporal ridge above mentioned. Due to the extreme thickness of the bone at this point, added difficulty was encountered in locating the antrum. The lateral sinus was exposed more by erosion than by efforts on my part. A thin trickle of pus welled up from beneath its bony covering and forced exposure downward. Again, below the knee a self-dissected cavity was encountered in direct contact with the jugular bulb. This abscess cavity contained frank pus and granulation tissue. It was fully exposed in all directions, thoroughly cleansed and packed with iodoform gauze. There was nothing eventful in the postoperative course. The first dressing took place on the fourth day and thereafter every second day. Prompt recovery followed without complications.

SUMMARY.

1. Two consecutive mastoidectomies revealed complicating peribulbar abscesses.

2. Considering the brief illness, the bone destruction in each case was unusually extensive.

3. Neither case could conceivably have recovered without operation. As it was, Nature was kind to have delayed more serious blood infection.

4. Smears of pus revealed the streptococcus as the offending organism. The species was not isolated. In view of the widespread destruction, one might hazard a guess in favor of the streptococcus mucosus capsulatus.

5. Recovery after operation was uncomplicated and uneventful.

222 Michigan Street.

A POSTAURICULAR FOREIGN BODY (A FEATHER) SIMULATING AN ACUTE MASTOIDITIS.

DR. GEORGE O. CUMMINGS, Portland, Me.

D. C. L., age 1 year, was referred to me on Dec. 21, 1930, because of postauricular swelling and redness.

His illness began on Dec. 15, when he began to carry an elevated temperature and to refuse to lie on his right side. On Dec. 17 the lymph nodes below his right ear were noticed to be enlarged and there was redness and swelling in the mastoid region. Throughout his illness he slept poorly and carried an average temperature of 103° at night.

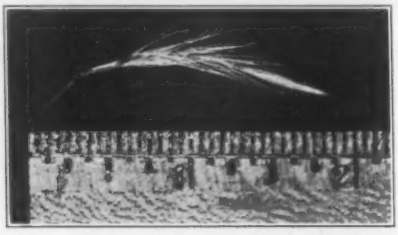
On Dec. 21 there was redness and swelling in the right mastoid region and the ear projected from the head. On the posterior surface of the auricle there was a small fester. The anterior surface of the auricle was normal, as were the eardrum and ear canal. The nose, throat and general physical examination were essentially negative. In view of the normal eardrum, absence of history of aural discharge and upper respiratory infection, and the presence of the

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication Feb. 3, 1931.

fester behind the auricle, a diagnosis of postauricular cellulitis of unknown origin was made.

The fester was opened and a long, thin, flexible object began to appear from the wound. This was gradually withdrawn and was at first thought to be a porcupine quill, which it resembled in size, but proved to be a black and white feather. The quill end was beginning to ulcerate through the skin.

I am absolutely unable to explain how the feather got into the child, nor could the parents, who are above the average in mentality.



It does not seem possible that a thin feather, about 2 inches long, could work its way through the skin, feather end first. There was no evidence that an entry had been made through either the anterior surface of the auricle or the ear canal. The apparent direction from which the feather was withdrawn would lead one to believe that its internal and feather end lay in the region of the angle of the jaw. It is unlikely that it could have worked through the Eustachian tube. Could it have worked out from the throat?

47 Deering Street.

FRACTURE OF THE NOSE—A PLEA FOR EARLY TREATMENT.*

Dr. Francis W. White, New York.

For such a prominent member of society the assaulted nose has received injustice rather than justice in the past.

The bony framework of the nose is subject to fracturing, as are bony structures elsewhere in the body. The classification is, in general, similar, but fractures are more frequently compound. The lower third of the nasal bone is the most common site of fracture. One point of marked difference is the result of a fracturing force usually leaving the broken bone where it is found by the examiner; whereas, in other parts endowed with greater musculature, the existing deformity may be caused by the action of the musculature, *e. g.*, humerus, femur. There are three logical directions for the fracturing force to travel (sometimes combined, however), namely, lateral, directly from in front, or, from, below. It is almost inconceivable a force could come from behind.

Our modern civilization has put a greater tax on one's dexterity to avoid accident, due to increased rapidity of transportation. This obtains particularly for children. They have been crowded off almost all vehicular lanes. Games are played and various forms of mechanical locomotion are attempted on crowded streets. Fracture of the nose in children and young adults is becoming more common. Fortunately, more attention is being given early to these cases, with consequent lessening of bad after-effects, both cosmetically and physiologically. For parents or others to neglect obtaining expert advice as quickly as possible after an injury to a child's nose is decidedly derelict on their parts. The old idea of staunching the flow of blood, putting a pad of gauze held down tightly over the injured nose by means of adhesive tape, is and should be relegated to the limbo of treatments by blood-letting for colds, cancers and corruptions. It should not obtain today. What then shall we do? We cannot expect every person with a suddenly fractured nose to rush immediately to a specialist. If this could be arranged it might obviate more drastic measures later. I feel one skilled in the use of the head mirror is in

*Read before the New York Academy of Medicine, Section of Laryngology and Rhinology, Nov. 21, 1930.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication Jan. 30, 1931.

a more logical position to handle the deformities associated with such a fracture than one not accustomed to its use.

Diagnosis: Pain, swelling, nosebleed. Manifestly, the history of the case is important. Even a layman can make a distinction between a concavity on one side of the nose and a convexity on the other. No displacement may occur at all, however. Beware of crepitus—it may be air or bone, or a combination of these. Not infrequently, it may be elicited by very gentle pressure, whereas strong pressure may mask it. It may be present one moment and absent the next. A dislocation of a nasal bone at its line of suture is similar to a fracture and may present almost identical symptoms. A constant tearing is indicative of lachrymal duct involvement and points to a fracture of the nasal process of the superior maxilla of the corresponding side. The X-ray may be helpful or not. Small films, if too strong a tube is not used, are helpful or absolutely positive. Inspection, especially *intranasally,* is important. A mobile fracture is not difficult to diagnose. An upper lateral cartilage may be torn free from its attachments, thus simulating a fracture. If in doubt, treat all as fractures as less harm can thus result, or, if seen late, await recession of the swelling, which may be hastened by appropriate means. A positive diagnosis may be made only after considerable manipulation has taken place under an anesthetic. When the nasal septum is fractured, it is usually in the posterior two-thirds of the cartilaginous portion or anterior half of the bony structure. It is possible to involve a part of both. Rarely, however, does a fracture of the nasal septum reach the posterior nares. There may be no evidence of a fracture, but at some later date a divided ridge of thickened tissue may be discovered corresponding to the line of healing. Most all of us have met with this condition at times when doing a submucous resection of the nasal septum in adults. The fracture of the septum may be either horizontal or vertical, the former being the more common. Distinguish fracture of the septum from dislocation of the septum.

Treatment: Reduce the fracture immediately. The longer the interval intervening between fracturing and setting the greater the force necessary to reduce. This may not necessitate the use of an anesthetic but the work must be done deftly. Unilateral cases can often be quickly and almost painlessly reduced by means of a flat, blunt elevator or one arm of the Adams' forceps. In fact, most any noncutting or specially designed reduction instrument may be used. If an anesthetic is required a choice may be made of a local or a general. If the latter is chosen, gas-oxygen is very convenient, but ether is a more certain method.

Personally, I do not favor the use of a postnasal plug for the control of hemorrhage—which is impractical under gas-oxygen anesthesia. Hemorrhage usually ceases spontaneously. If it does not, then fracture of the skull must be considered. Light gauze packing intranasally, but posteriorly to the site of manipulations and secured by a strong, silk suture which hangs outside the nostril, will suffice if ether is administered and there is considerable bleeding. Instrumentation for reduction is as noted above, or one arm of the Adams' forceps may be placed inside the nose and the other, protected by a small piece of rubber tubing, on the outside of the nose and the fractured bone lifted bodily into place. One thing is necessary, that is over-correction, so that the bone may be easily slipped back again into place, and remain there without stress or strain. Swelling may or may not be marked by this time but, personally, I have never had untoward results therefrom. Infrequently, a hematoma of the septum or lateral wall may occur. As the weight of the osseous structures involved is little, and there is but little or no muscular traction, the main cause of properly set nasal bones not remaining in place is the exerting of internal or external force, such as violent blowing of the nose or violent sneezing, rough manipulation or accidentally hitting the nose. A sore thumb is somewhat of a parallel in the latter instance—it is always being knocked! Skin cuts are sutured if necessary under strictly surgical technique.

The writer has used or seen used most of the mechanical devices recommended as retention dressings with the result that either nothing is used or recourse to one of the three following forms of dressings is resorted to, namely:

1. A butterfly pattern of the nose and cheeks is cut out of a No. 20 gauge copper wire mesh. This is lined with light, surgical lint, placed over the bridge of the nose and cheeks and held in place by means of collodion painted to the outside of the splint, or one of the liquid adhesive fluids may be used. Sutures may be attached to intranasal splints and passed through the dorsum of the nose and secured to the mesh; or,

2. Surgical lint cut as above, the surface to come in apposition to the bridge of the nose and cheeks, is coated with one of the liquid adhesives and then applied and held in place until it dries; or,

3. A thin layer of cotton, in form as above described and placed as above noted, is then held in place by applying collodion and allowing this to soak through to the skin.

Any of the above dressings will hold firmly for five or six days if properly applied and new ones substituted as required. The skin

is very tolerant. The splints combine strength, rigidity, compactness and cleanliness. They are easy to put on or take off and do not add weight over the newly-set bones. Blowing of the nose is absolutely forbidden. Lead, copper or other materials may be shaped to the nose, to be worn as a noseguard during sleeping hours after the second week. Usually firm union is obtained in twelve days to three weeks.

Regarding intranasal packing or support. It is rarely much pressure is required; in fact, poor results may be obtained by forcing the bones outward to an inordinate degree. Usually, however, the intranasal swelling is such that the mucosa of the turbinates, lateral wall and septum is so marked that a natural splint is formed. However, if much space is seen on intranasal inspection, a thin piece of *dry* Bernay splint is inserted, moistened and left *in situ* for two to three days, a few drops of one of the mild antiseptics being instilled once or twice daily for cleansing purposes. This procedure may be repeated as found necessary. It is rare that cumbersome extra or intranasal appliances are required after recent reductions.

Contraindications: Profound surgical shock; concussion of the brain; possible fracture of skull (one end of a nasal bone may enter the anterior fossa); extensive badly infected trauma with compound comminuated fracture; possibility of traumatic erysipelas being present; excessive emphysema; compound intranasal and acute suppurative sinusitis.

Complications: All as above noted; also abscess formation, bone necrosis and liquefaction of cartilage.

174 West 58th Street.

TWO CASES OF INFECTIOUS MONONUCLEOSIS.*

DR. WALTER D. CHASE, Bethlehem, Pa.

Many years ago Pfeiffer described a disease characterized by fever, enlarged lymph nodes, slight redness of the throat, etc., which affected chiefly children, and which he termed "Glandular Fever."

At the Detroit meeting of the American Academy of Ophthalmology and Otolaryngology, in 1927, one of the teachers on laboratory work in connection with ear, nose and throat diseases, made mention of a newly described disease, termed "Infectious Mononucleosis." He warned us to be on the lookout for this condition, since the throat symptoms, which are among the earliest features of the affection, bring the patients to the rhinolaryngologist. Not until this winter have I had the good fortune to diagnose one of these cases, though a case or two may have slipped through my hands, since not every possible case one sees in the office is given a differential white cell count, and it is this differential count which furnishes the chief diagnostic aid.

The Pfeiffer[1] malady occurred mainly in children. No blood examinations of his cases are believed to have been made. Infectious mononucleosis occurs in "young adults, mostly males," and there is a distinctive blood picture always present. Therefore, those claiming the two diseases one and the same are not provenly correct and it is doubtful if such is the case.

Charles E. Connor reviews the disease very concisely, bringing out the following facts: Infectious mononucleosis is announced by the symptoms of an acute infection, *e.g.*, headache, fever, chills, sore throat and malaise. The throat condition is constantly present, such as intense injection of faucies, scattered patches of exudate or actual ulcerative lesions on the tonsils. General adenopathy, most marked in the upper cervical region, but involving also the axillary, inguinal and epitrochlear lymph nodes, is one of the most conspicuous features. The spleen is frequently enlarged, sometimes the liver also. Petechiae may be seen on the buccal surfaces. Cough may be present, possibly due to enlarged mediastinal nodes. The fever may go as high as 102° or 103°, but often very slight elevations are noted. Abdominal pain is sometimes complained of. The enlarged lymph

*Reported at Staff Conference, St. Luke's Hospital, Dec. 15, 1930.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication Feb. 5, 1931.

nodes are smooth, discrete, tender to a slight extent, firm and may reach a diameter of 1 or 2 c.m. The hemoglobin is normal, but the white cells are markedly changed, this being our chief diagnostic feature. The leukocytes increase shortly after the onset and the mononucleosis is present by the end of the first week. The leukocytosis ranges between 11,000 and 26,000, with a marked lymphocytosis, the lymphocyte percentage being between 57 and 92 per cent. The red cells are unchanged.

Warfield T. Longscope has written one of the best articles on this disease, published in the December, 1922, issue of the *American Journal of the Medical Sciences.* He describes three types of these mononuclear cells: *1.* A small mononuclear, identical with the small lymphocyte; *2.* large mononuclear cells, looking like the large mononuclear cells of normal blood; *3.* mononuclear cells of the type not found in normal blood, namely, a cell somewhat larger than the normal lymphocyte, the nucleus being oval, kidney-shaped or lobulated and staining deeply with the dyes of the Romanowski series, the cytoplasm being basophilic, nongranular and not giving the oxydase reaction.

The symptoms usually terminate within three weeks, though the lymph nodes may remain enlarged for several weeks.

Diagnosis: The *onset* makes very easy the mistaking of the disease for influenza, tonsillitis or typhoid fever. At the *height* of the disease, other conditions having general glandular enlargement must be excluded, such as diffuse tuberculous lymphadenitis, Hodgkin's disease and leukemia. Influenza is a term very much used by the well nourished, over-confident physician; it covers a multitude of his sins. After the patient has done his part of the job by telling his doctor why he called him and by answering his questions, the modern public actually expects its doctor to do some more or less scientific investigating. I need not describe all the steps in detail, but permit me to use again my favorite dogma: Every doctor may become a really great physician by "working hard on his cases." The child who is born with average brains, but who is not cursed with lack of ability for hard work or an over-supply of conceit, can become the "great" physician I have described, because we have been told, and many of us have seen it proven, that a "genius" is merely one who has so trained his brains that they really work when the possessor is going through the motions of working. Such a man will not take a look into a throat and announce that the case has "tonsillitis." He will look, feel, percuss and listen—use his cortex, remembering that the practice of medicine is really a serious calling, and the super-

ficial appearance of the patient's pharynx will not prevent his completing the investigation and arriving at a correct diagnosis. The other diseases mentioned must be ruled out in the same way, by wide-awake physical examination and the intelligent use of the laboratory. Lymphatic leukemia has a much higher white count, often 50,000 by the time the angina appears; later, very much higher, and the cells are immature lymphoblastic type, instead of the mature but abnormal mononuclear cells of infectious mononucleosis. There is also marked anemia and soon a hemorrhagic diathesis in leukemia.

Case 1: Male, white, age 18 years. Referred by J. F. Connaly, D. D. S., Oct. 17, 1930, because of the noticeable cervical adenitis. Patient stated he had been somewhat ill for eleven days. First had what he called "headache in the eyes," which made reading blueprints difficult. Sore throat two days previously. On the day examined, pain below the right mastoid. Chilly one day last week. I found the temperature 99.3°, pulse 88, it being evening observation. Marked enlargement of cervical lymph nodes, which felt deeply situated, the greater swelling being on the right side. Tonsils congested and there was a small area appearing as a slight ulceration on the anterior border of right. On soft palate, fine petechiae. Blood examination by Dr. Haig showed the following: Hemoglobin, 100 per cent; W. B. C., 20,000; neutrophilic polymorphonuclears, 13 per cent; eosinophiles, 1 per cent; small lymphocytes, 24 per cent; large lymphocytes, 51 per cent; monocytes, 11 per cent. "Blood picture is that of infectious mononucleosis," stated Dr. Haig. I had told him of my belief that we had one of the cases for which I had been on the lookout, and so it turned out to be. The patient's physician did not give me any further information on the later phases of the case, but his dentist has seen him and states that he is now well.

Case 2: Male, white, age 20 years. Student at Lehigh University. Referred, Oct. 21, 1930, by his physician for tonsillectomy. Dr. A. R. Zack, attending physician, stated patient had been ill several days, at one time being so constipated that he had to be sent to hospital for administration of enemas. When patient reported for examination and the arranging for operation, questioning brought out the history that he had continued attending classes, though he could not accomplish much. He had had, besides the constipation, frontal headache, malaise, sore throat, etc. I found injected pharynx, enlarged cervical lymph nodes, especially that of the left "tonsillar node." I advised his entering hospital for blood examination, postponing preparations for tonsillectomy until other things than tonsil infection could be ruled out. Dr. Haig's report follows: W. B. C.,

17,000; polymorphonuclears, 12 per cent; lymphocytes, 77 per cent; monocytes, 11 per cent. Later reports from a laboratory in Newark follow: Oct. 25, four days after first count, W. B. C., 15,450; polymorphonuclears, 9 per cent; lymphocytes, 53 per cent; monocytes, 6 per cent; myeloblasts, 23 per cent; lymphoblasts, 9 per cent. On Nov. 5, W. B. C., 11,100; polymorphonuclears, 36 per cent; lymphocytes, 60.5 per cent; monocytes, 2 per cent; lymphoblasts, 1.5 per cent. On Nov. 18, W. B. C., 15,300; polymorphonuclears, 60 per cent; lymphocytes, 35 per cent, monocytes, 5 per cent. This patient had gone home, where good laboratory observations were conducted.

Having the presentiment that some of these cases might fall into the hands of over-eager laryngologists, who do tonsillectomy sometimes without sufficient indications, I wrote Dr. Crowe, of Johns Hopkins; Dr. Coates and Dr. Fetterolf, of the University of Pennsylvania; Dr. Fenton, University of Oregon; Dr. Beck, University of Illinois Medical School; Dr. Dean, Washington University Medical School, and Dr. Canfield, University of Michigan. Each and every one of them advised against tonsillectomy during the period of abnormal blood picture. This advice, given by men of rich experience and responsible position, should be our guide. If, after more investigation of this disease has been made, we change our minds, then we can operate at another time of the disease's course.

Treatment consists in symptomatic measures, there being no specific known at present. After the patient has satisfactorily improved, tonsillectomy can be considered; when good judgment *dictates*, tonsillectomy.

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230 East Broad Street.

RETROPHARYNGEAL ABSCESS WITH SEPTICEMIA AND RECOVERY.

Dr. Charles N. Gettes, Boston.

The following case is that of a male child, H. B., age 4 years, whom I saw in consultation on April 10 because of a spontaneous throat hemorrhage, which was shortly afterwards followed by the vomiting of some bright red blood and what looked like a predigested piece of chicken liver clot.

The history I elicited from the attending physician was that of a sore throat, an acute follicular tonsillitis, three weeks prior to this date, which under routine care subsided, enabling the child to resume his normal habits. Ten days ago the patient developed a right, anterior, cervical adenitis, and because of a slight degree of rise in temperature was put to bed. Hot poultices were ordered for the swollen cervical region.

At no time up to now was there any peculiarity of voice, aphonia or stridor. However, the child progressively lost ground, despite the fact that the cervical swelling had subsided considerably and was no longer tender to touch. Sinc April 4 the mother states that the patient has been quite listless and has had alternating chills and flushes of warmth. The temperature readings ranged from 99° F. in the morning to 106° F. during the evening.

Finger palpation at varying frequent intervals, and examination of the throat under direct vision failed to reveal any pharyngeal bulging, supratonsillar or peritonsillar swelling.

Physical examination at the time of consultation was as follows: A pale, emaciated little boy, of about 4 years of age, is seen lying in bed, with no particular stridor of any kind. He is febrile and shows a tendency toward somnolence, rolling his eyes up and feigning sleep as one approaches his bedside. He is quiet and assumes no particular position that indicates pain anywhere in the body. The temperature is 103° F., pulse 140, respiration 36.

Head: Macrocephalic, pale forehead with visible distended venules. He is not perspiring.

Eyes: Pupils are equal and react readily to light and accommodation. The ocular movements are good in all directions. No ptoses are noted.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication Jan. 31, 1931.

Ears: Essentially negative, both membrana tympani being normal in every detail.

Nose: A mucopurulent secretion is present in both nares, filling the respiratory fissures. The middle meati are clear, as are also the olfactory fissures. The nasal mucosa is slightly injected.

Throat: About one-half c.m. to the right of the median line of the oropharynx and just under the soft palate is a bleeding vein, which was produced by the spontaneous rupture of what must have been a parapharyngeal or retropharyngeal abscess. It is a venous ooze and was readily controlled by a hemostat. Both tonsils appear injected but are neither edematous nor ballooned out with fluid.

Larynx: Normal; airway good. No abnormalities noted, either on phonation or on respiration.

Neck: Marked adenopathy of right anterior cervical chain. The glands are not tender to touch, but are very firm to the palpating finger.

Heart and Lungs (examination by attending pediatrist): No abnormalities are noted on percussion or auscultation.

Abdomen: Soft, not distended. Superficial venous markings are readily seen. The spleen and kidneys cannot be palpated.

Extremities: The patellar reflexes are somewhat exaggerated. Kernig sign is negative. The Babinski, Oppenheim and Brudzinski signs are all negative.

The patient was admitted to the Beth Israel Hospital on April 11, where a bulging mass in the right lateral oropharynx was incised and sucked clean of blood and pus. This incision was connected with the point of spontaneous rupture by means of a Kelly hemostat.

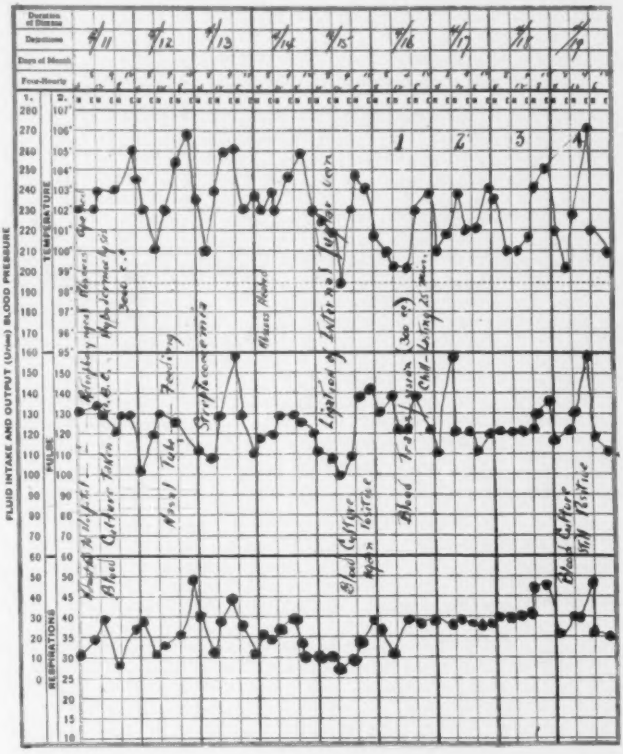
A blood culture was taken from the median basilic vein and a complete blood study was made. X-rays of the chest and cervical spine were also taken, but with negative findings.

The blood picture was that of a marked secondary anemia and a poor resistance, there being only 10,000 W. B. C., with a fairly normal differential count. Blood culture after 24 hours was negative.

The child was fed through a nasal tube, since he was unable to take nourishment. Three thousand c.c.m. of glucose and 5 per cent sodium bicarbonate solution were administered subcutaneously. The pulse and temperature curves continued septic.

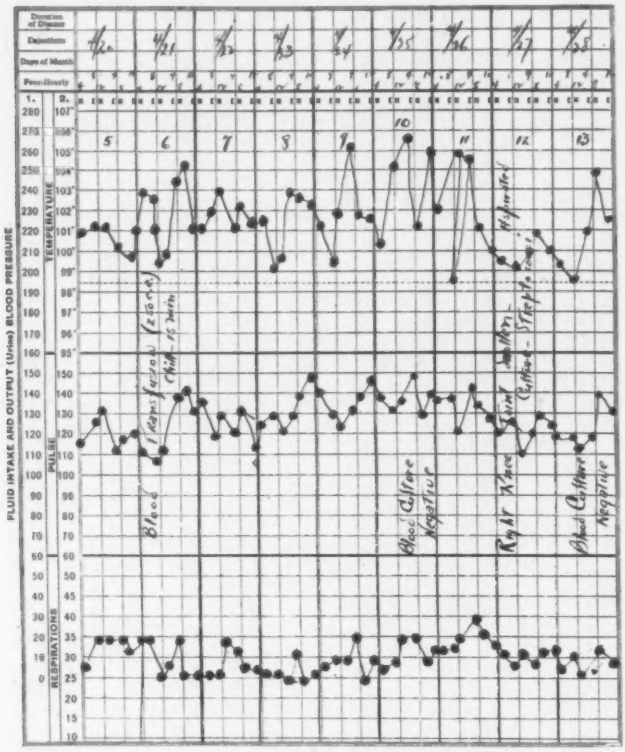
After 48 hours an undifferentiated streptococcus was recovered from the blood stream. Another complete blood count revealed a hemoglobin of 55 per cent; red cells, 2,600,000; white cells, 20,000; with 80 per cent polymorphonuclear neutrophiles and only 6 per cent of immature cells as differentiated by the Schilling index.

The temperature curve was still of the distinct septic type, as is seen in cases of lateral sinus thrombosis, with or without septicemia. The morning remissions and evening rise in temperature, the chills and sweats remaining so significant, that exploration of the internal jugular vein presented itself as the next most logical step in the treatment of this patient.



The following morning the child's condition having so markedly improved under gas-oxygen anesthesia, the right internal jugular vein was explored. The jugular vein was normal in color and showed no evidence of thrombosis. It was ligated *in situ* at the level of the median portion of the inferior belly of the omohyoid muscle. A chain of densely matted-together lymph nodes, which appeared as though ready to undergo necrosis, was dissected from the neck. This neces-

sitated going somewhat lateralwards. On tracing the chain it was found to extend down to as far as the clavicle. At this point it was ligated and then removed above the tie. Troublesome bleeding was encountered, requiring ligature for control. Blood was withdrawn from the jugular vein for culture.



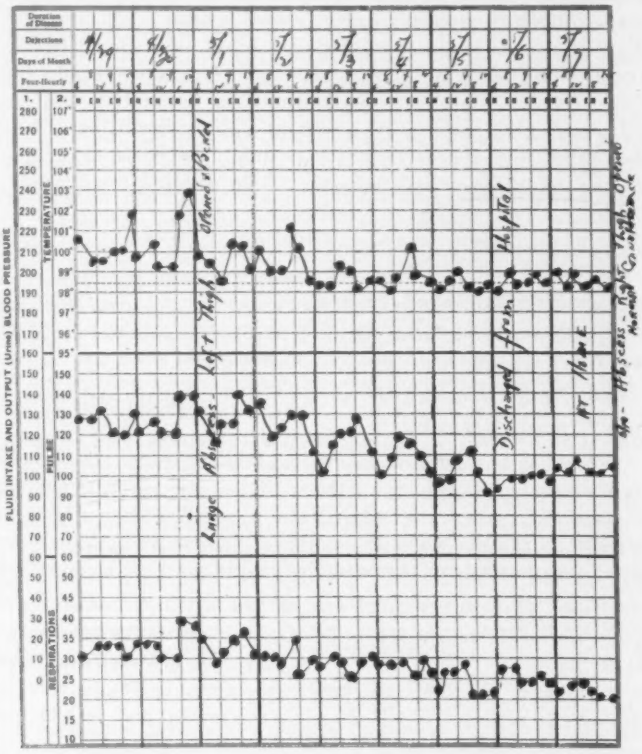
Shortly after the child had recovered from the effects of the anesthesia, a ptosis of the upper eyelid, a myosis of the right pupil, and an enophthalmus was noted (Horner's syndrome).

After 24 hours blood culture, taken from the internal jugular vein at the time of operation, was positive for the anhemolytic streptococcus.

The temperature dropped to 103° F. and the child seemed somewhat improved. Two successive blood transfusions, at four-day inter-

vals, using a Type 4 donor, were productive of steady improvement in the patient's general condition.

Microscopic study of the removed glands revealed several fragments of lymph nodes and of fat and fibrous tissue. The lymph nodes showed a normal architecture and the follicles and sinuses were easily made out. Many of the sinuses were stuffed with lympho-



cytes. Throughout, the germinal centres were large and active. The reticuloendothelial cells everywhere were swollen and vacuolated. These vacuolated cells could be found in the pulp and also lining definite sinuses. The capsules of the lymph nodes were definite and intact in most places and not invaded by lymphocytes. There were no polymorphonuclear leukocytes and no areas of necrosis in the glands.

The changes here, although rather diffuse, are slight, the structure of the lymph node being well preserved, thus giving numerous clear-cut pictures of reticuloendothelial cells still *in situ* in the sinuses and showing hyperplastic changes.

Such a picture is present either in the peripheral node of a chain draining an area where toxic material is being generated, or in a position where a rather weak toxic material is coming to the lymph nodes through the lymph channels. Such a node might progress in either of two directions. A continued influx of toxic material, or invasion with bacteria, as usually would soon occur, would cause small areas of necrosis, polymorphonuclear infiltration and some fibrin, and might eventually go on to abscess formation. On the other hand, since such a node is very little changed from normal, a cessation of the deleterious influence would permit a rapid return to normal[1].

Locally the abscess was entirely healed.

A third blood culture, taken on April 19, was still positive for the anhemolytic streptococcus. A blood count on this date showed a hemoglobin of 60 per cent; R. B. C., 3,630,000; W. B. C., 18,500, and 80 per cent polymorphonuclear neutrophiles.

Blood culture repeated on April 25 was negative, and again on April 27 was still negative.

The right knee joint now became swollen and tender and the child was unable to move his knee because of the pain. Needle puncture and aspiration were instituted and this was followed by fixation of the joint, using a molded cast. Culture of the removed joint contents showed the same type of organism that had invaded the blood stream.

On April 26 the temperature peak settled to 101° F. and the child rapidly showed a return to normalcy. On May 1 the temperature was normal and remained so. A large mass was observed on the left lateral aspect of the thigh, and under gas-oxygen anesthesia this was explored. A large walled-off abscess was emptied and packed with vaselin gauze. Culture again showed the anhemolytic streptococcus.

The temperature still being normal, the child was sent home, with a right Horner's syndrome, a stiff right knee joint, and a healing abscess of the left lateral thigh region.

At home he soon regained the use of his right knee, the left thigh abscess healed. Soon after this, a swelling was noticed over a similar location on the right thigh. This was cut into, disclosing another walled-off abscess. It was treated in a like manner.

Ten weeks after his admission to the hospital the child was discharged, perfectly well. The right Horner's syndrome had cleared up entirely, and the boy was able to run around and play.

CONCLUSIONS.

The internal jugular ligation was here performed as a therapeutic measure, on the same principle that it is done in cases of mastoiditis that are complicated by lateral sinus thrombosis and septicemia, or mastoiditis and septicemia. The pharyngeal plexus of veins which drain the neck spaces in the oropharynx are just as liable to undergo thrombosis, or act as carriers of the septic materials into the blood stream.

Despite the fact that the causative organism was the anhemolytic streptococcus, and that the usual methods of combatting septicemia, *viz.*, forcing fluids and repeated blood transfusion, were employed, I feel that had the vein not been investigated, and a thrombosis existed, our ordinary methods of treatment would have been of no avail. Similarly, the ligation *in situ* was performed to prevent the direct influx of the septic-laden blood contained in the pharyngeal venous plexus, into the major blood stream; and to render the vein impermeable to its surroundings.

Whether the source of infection was through the angina (which is probable), or through the lymph nodes (which, as a rule, is not very probable), the ligation should certainly be considered early when spontaneous rupture and surgical drainage of the abscess fail to relieve the attendant symptoms. With this thought in mind also, the exploration was undertaken, for locally the abscess had healed.

The Horner's syndrome was undoubtedly due to having included in one of our ligatures a filament of the ciliospinal centre. After absorption of the ligature, the injured nerve gradually recovered, with ultimate clearance of the right upper lid ptosis, the pupillary myosis and enophthalmus.

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371 Commonwealth Avenue.

SECONDARY TONSILLAR HEMORRHAGE.*

Dr. James H. Earley, Washington.

Secondary tonsillar hemorrhage has long been the problem and cause of many a sleepless night for the otolaryngologist and I dare say every surgeon has had experience with it one or more times. Dabney[1] states "as long as the operation of tonsillectomy is performed, whatever be the method, just so long must the hemorrhage, immediate or remote, accompanying the procedure be a matter of prime importanec to the conscientious laryngologist." Secondary tonsillar hemorrhage in this paper refers to that which occurs from one to 14 days after the removal of the tonsils. It is not as rare as one might be led to believe, though secondary tonsillar hemorrhage occurring from the fifth to the fourteenth day is rare.

The day of tonsillotomy is past, and justly so, for in its place has come tonsillectomy, or enucleation of the entire tonsil, with its so-called capsule and the consequent removal of all diseased tissue. This operation should be considered a major surgical procedure and should always be done in the hospital. The cause of secondary tonsillar hemorrhage is often due to faulty technique in operation, such as incomplete removal, cutting into the underlying muscles and removal of one or both pillars.

Great care should be exercised in the operation because of the rich blood supply of the tonsil and an effort should be made to save the plica triangularis, because at this point at the angle of the mandible lies superficially the external maxillary artery and a little further away the internal and external carotid arteries, which may be incised if the tissue is cut too deeply.

We should institute such proper preventive measures, good operative technique and proper postoperative treatment in order to prevent secondary tonsillar hemorrhage. In spite of the fact that these measures have been taken, secondary tonsillar hemorrhage does occur. Secondary tonsillar hemorrhage is not due to minor differences in technique. Bleeding will occur no matter what method is employed, since the particular method will not alter the chances of hemorrhage, and it also will occur under local or general anesthesia; at least this has been my experience. However, my best results have been

*Thesis submitted to the Faculty of Otolaryngology of the Graduate School of Medicine of the University of Pennsylvania in partial fulfillment of the requirements for the degree of Master of Medical Science (M.Sc. Med.) for graduate work in otolaryngology.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication March 4, 1931.

obtained in the LaForce method of tonsillectomy used in the Coates-Skillern Clinics at the Post-Graduate Hospital of the University of Pennsylvania. I find this method can be used in 99 per cent of my cases, both under local and general anesthesia, providing the patient has not already had an incomplete tonsillectomy. Skillern[2] reports not having had a single secondary hemorrhage in 12 years by the use of the LaForce method. I have had three in a series of three hundred cases.

One should be on guard for hemophilia, which is a common cause for bleeding, although seldom met with. However, despite a negative history, the coagulation and bleeding time should be taken and I am in the habit of giving some remedy to improve the coagulation of the blood, such as calcium lactate by mouth, calcium chlorid intravenously, or coagulin, thromboplastin or hemoplastin intramuscularly several hours before the operation if the clotting or bleeding time is five minutes or over. Cases having a normal clotting or bleeding time before operation may have a deficient clotting or bleeding time after operation because of the large amount of blood lost at the time of operation and the consequent decrease in the volume of blood, which may result in secondary tonsillar hemorrhage. I have tested the clotting time of several cases that bled profusely at the time of the operation and in each instance I found the clotting time to be increased after operation. In cases which bleed profusely at operation I give large doses of glucose and soda by rectum and soda by mouth as soon as nausea and vomiting has ceased to prevent any acidosis because secondary hemorrhage is always threatened in these cases, as they have a prolonged clotting time. That diet alters clotting time has been brought out experimentally in animals by Kugelmass[3], *et al.* He has proved that the tendency to bleed or clot is definitely influenced by diet.

Physical examination may reveal disease of the kidney, heart, lungs or circulatory system. It is definitely known that such diseases predispose to bleeding and I shall include a case to illustrate this point. Papadopoulos[4] reports a case of a 15-year-old boy with congenital heart disease (apparently a patent foramen ovale), who had periodic attacks of bleeding from the surfaces of the tonsils and postnasal pharynx. Tonsillectomy resulted in dangerous complications, but since recovery he has been completely free from all nose and throat symptoms.

In children, Roentgenologic study for persistent thymus gland is recommended in case a general anesthetic is to be used. An enlarged thymus may cause constriction and displacement of the trachea. We

have such a case at present in the Children's Hospital. X-ray therapy reduces the size of this gland very rapidly.

Patients having recently suffered from an acute inflammatory condition of the throat, no matter what the cause, should not be operated upon for several weeks after recovery. Inflammatory tissues have an abnormal supply of blood, and consequently there is increased danger of bleeding and infection with ulceration of tissues, which may involve an open blood vessel and cause premature separation of the slough (Dabney[5]).

As stated by Nothenberg[6], in cases of diseased tonsils there is also a degenerated condition of the muscles in the neighborhood of the tonsils and the muscles are soft and friable. An artery forceps goes through the tissues as if it were cheese and it will not hold the ligature.

A good many secondary tonsillar hemorrhages are, no doubt, due to secondary infection, which causes sloughing of the vessel before the intima is fully sealed. I wish to report a case of this character here. White[7] reported seven cases which caused a slough of the vessels before the intima had become fully sealed. Tibbets[8] reported five cases of secondary tonsillar hemorrhage due to Vincent's organisms. Smith[9] reports a very interesting case of gas bacillus infection following tonsillectomy, which, while not complicated by hemorrhage, resulted in death.

One cause of secondary tonsillar hemorrhage is over-exertion, such as exercise, laughing, screaming and over-use of tobacco and alcohol, which may open a thrombosed vessel. A case of this kind is reported here.

Many surgeons, among them Tilley[10] and Wells[11], believe in the routine ligation of all tonsillar vessels and have adapted special instruments and technique for suturing, but even after this procedure, secondary hemorrhage has occurred. Wells[11] sutures two and sometimes as many as four points where bleeding is likely to occur.

When secondary hemorrhage does occur one should particularly note the upper part of the fossa, because at this junction the largest blood vessel which supplies the tonsils enters the fossa. One should be careful that all bleeding areas are stopped before the patient leaves the operating room and a simple ligature should be done, because the use of a needle in the throat is a dangerous practice, as the needle may break or drop off the catgut or silk and be lost. Recently such a case came under my observation, in which the resident interne broke off a needle in the soft tissue. I was notified of this and we recovered the needle the following day. Ligatures or sutures that are taken should be removed within 12 to 24 hours, as

thrombosis certainly will have occurred in that period of time and removing them at this time will prevent the formation of adhesions, slough and deformity.

Unnecessary use of sponges in the throat and tonsillar fossa should be avoided, as they often tend to defeat their own purpose and to increase, rather than lessen, the liability to bleed, especially at the time of the operation. They should be used gently in the throat to blot the blood.

Secondary tonsillar hemorrhage can be controlled by many methods. Oftentimes it will stop by the simple method of pressure or the placing of a sponge in the particular tonsillar fossa that is bleeding. Styptics, such as antipyrin, silver nitrate, peroxid, dioxygen, Monsel's solution, tincture of ferric chlorid or glycerit of tannin, can be used on these sponges, but I find that a plain sponge acts just as well as a sponge with any of these styptics on it if the sponge is going to stop the hemorrhage at all. The simple injection of 1 per cent procain epinephrin often stops oozing areas without recurrence, providing the patient is not suffering from hypertension. All patients should be given sedatives and an ice bag applied to the neck, and they should be kept quietly in bed. I have found the use of coagulins intravenously and intramuscularly of great help, but I have been disappointed in their results when given by mouth. Ligatures should be applied where there is active bleeding if the bleeding point can be found. Sutures should be used only as a last resort. Some operators like to suture a sponge in the fossa when there is generalized oozing. Thermocautery has been used to stop these hemorrhages, but I have had no experience in the results of this method.

Most important of all is the proper postoperative care of the patient and sometimes little stress is placed on this significant point in the prevention of secondary tonsillar hemorrhage.

REPORT OF CASES.

Case 1: About three years ago I had my first tonsillar hemorrhage. In this case the cause was quite apparent. This occurred in a female patient of about 42 years of age, in whom I did a secondary tonsillectomy. She came to me complaining of difficulty in breathing through the nose, pain over the right cheek and sore throat. She also complained of swelling of hands and feet. Transillumination of her sinuses revealed a positive right maxillary sinusitis. Large pieces of tonsil were noted in each fossa and pus could be expressed from them when pressure was applied. Her blood pressure was 185 systolic and 90 diastolic. Examination of her urine revealed many pus cells and a few hyalin and waxy casts. Coagulation time and bleeding time were normal. After her sinus condition cleared,

I removed her tonsils under local anesthetic (1 per cent procain and epinephrin), using the sharp dissection method because of the large amount of scar tissue formation. Twenty-four hours later, as she was about to leave the hospital, she had a hemorrhage and since she gagged a great deal I thought I would try to control the hemorrhage by giving her some ceanothyn by mouth. One tablespoonful was used and repeated for six doses. The patient vomited the third and sixth dose and the bleeding seemed to become more active after these vomiting spells, so ceanothyn was discontinued and the bleeding point ligated. There was no further recurrence of bleeding. Hypertension and kidney infection were undoubtedly the cause.

Case 2: Some months later I had my next case, which was in a child of about 8 years of age. This child had been backward in school, anemic, under-nourished and had had frequent attacks of sore throat. He also presented adenoid facies. This patient was operated on under gas anesthesia, the LaForce method being used. There was no primary bleeding at the time of operation and after a stay of 24 hours in the hospital, he was discharged. Seven days after operation I was called to the home of this child by his mother, who said the child had been bleeding for two hours. As this case was a child, I made no attempt to stop the bleeding at home. He was sent into the hospital, where, under gas ether anesthesia, the bleeding point was noted in the upper left fossa in an eroded area, where a slough had separated. The bleeding point was clamped and a ligature applied. Culture of both fossae was made and a report came back, streptococcus hemolyticus predominating, with a few Vincent's bacillus. The patient was returned to the ward, where he received glucose and soda by rectum and some normal saline under the breast. The following day he was given an extract of spleen and bone marrow and dioxygen gargles. Fluids were forced and he improved rapidly and was discharged from the hospital five days after admission. This case was undoubtedly a case of secondary tonsillar hemorrhage caused by secondary infection.

Case 3: My third case of secondary tonsillar hemorrhage was in a healthy young girl of about 21 years, athletic in type and whose skin was bronze from outdoor life. She gave no history of bleeding and there was no bleeding at the time of operation. The coagulation time and bleeding time were within normal limits. The operation was performed under local anesthetic, using procain and epinephrin, 1 per cent. I used a LaForce tonsillectome. The patient was discharged from the hospital 24 hours after operation, feeling well and having no complaint. Three days after operation she reported to my office and her throat looked fine. Four days after operation she

went to a summer resort, where she smoked, danced and indulged in some alcoholic beverages. On the fifth day she returned home and she began to menstruate. That evening she had a profuse hemorrhage from the left fossa. The bleeding point was noted at the lower part of the fossa at the base of the tongue. Slight pressure with a sponge readily controlled the bleeding, and as the patient gagged a great deal, I simply allowed a string sponge to remain in her throat until the next morning. No bleeding occurred after this. I believe hemorrhage would not have occurred in this case if the patient had remained quietly at home.

CONCLUSIONS.

1. We should exercise the best preoperative and postoperative methods to prevent secondary tonsillar hemorrhage.

2. The LaForce method is by far the best method in the prevention of secondary tonsillar hemorrhage, as there is very little handling of the tissues and the hemostat can be left on as long as the operator desires.

3. The fact that the coagulation time and the bleeding time are within normal limits does not indicate that the patient will not bleed at the time of operation or have a secondary tonsillar hemorrhage.

4. We should adopt such measures for control as will be indicated by the particular case when hemorrhage does occur.

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THE PHARMACO-DYNAMICS OF LOCAL MUCOUS MEMBRANE MEDICATION.

DR. CARL SCHEFFEL, Miami, Fla.

While brevity is said to be the soul of wit, abstract terminology in scientific matters is apt to lead to misleading conclusions. The title of this paper refers more particularly to the mucous membrane of the upper respiratory tract; for it is obvious that physiologic, pathologic and, consequently, also therapeutic factors applying to the mucous membrane in one part of the body may materially differ from those in another.

The therapeutic objective sought with most forms of localized mucous membrane medication is: the production of an astringent effect, antipathogenisis ranging in degree from that of making the mucous membrane unsuitable soil for pathogenic growth and the absorption of pathogenic products, to that of producing complete destruction of living organisms, mechanical protection, or a combination of these effects.

Any therapeutic endeavor that does not take into consideration the physiology and pathology of the affected tissues and the pharmacodynamic action of the drugs used, as based on those factors, must be construed as empirical at its best. The modern tendency fortunately points towards more rational therapy based on a sound scientific background; therefore, let us primarily briefly review some of the physiology and pathology of this specific mucous membrane.

According to Halliburton[1], the greater portion of the nasal cavity is lined with epithelium of the ciliated variety, throughout which is distributed numerous mucus-secreting goblet cells. Also, as is well known, epithelium is nourished and bathed by lymph, which Halliburton[1] states is composed of about 90.3 per cent water, and the inorganic salts of which consist of about 75 per cent sodium chlorid. The mucin secreted by the goblet cells is readily precipitated by acetyl and other acid radicals, but is soluble in dilute alkalies.

The pathologic picture for which localized mucous membrane medication is commonly employed is almost without exception that produced by some form of an irritant upon the membrane. Schmaus[2] portrays this picture essentially as follows, from within towards the

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication Jan. 22, 1931.

surface: engorgement of the arterial capillaries and stasis of the venous capillaries, resulting in profuse transition from the vessels into the surrounding tissues. The irritated cells produce an excessive outpour of mucus containing broken down epithelial cells and mucus corpuscles.

In connection with the therapeutic considerations of any prescription intended for localized mucous membrane medication, such matters as insulation, viscosity, solubility, hydrogen ion concentration and the multiple effects of most drugs, become of the utmost practical importance. Let us, therefore, consider separately these pharmacodynamic points in the order named.

Primarily, no combination whose vehicle wholly or partially acts by insulating its contained active ingredients from the tissue they are intended to influence, can be deemed physiologically compatible to the degree required for rational therapeutics. Neither the anatomy, physiology nor chemistry of the mucous membrane discloses evidence tending to show that oily solutions so commonly used for nasal mucous membrane medication are suitable for such therapy. In fact, quite the opposite seems to be the case; namely, that they are physiologically and chemically incompatible with the mucous membrane. Physiologically, because they will not mix with the 90.3 per cent of water of which this membrane is composed, nor with the 75 per cent watery mucous it secretes. Chemically, they are incompatible because the fatty acids in the oils tend to precipitate the mucin secreted by the goblet cells.

The result of this physiologic and chemical incompatibility of oily solutions is the production of a more or less insulating effect between the active ingredients contained therein and the tissues intended to be affected thereby. This insulating effect of oil is primarily physical, and, as we shall learn later, secondarily also electrical in nature. Certainly, in every instance in which more than the mere mechanical protective properties of oils are desired, these insulating influences are highly detrimental from a therapeutic standpoint. Aqueous solutions or vehicles are practically free from these insulating drawbacks.

Moreover, A. Coolidge[3] directs attention to the fact that the principal function of the nose in man is to warm, moisten and filter the inspired air. Obviously, an oily solution applied to the nasal mucous membrane interferes with the rapid warming of the inspired air because of its insulating property; and it seems rational that the moistening of the inspired air was intended by Nature to be made with water, rather than oil. Then, too, although oil may act as an efficient filter, it has a strong tendency to clog the mucus-secret-

ing cells, due to its acid radicals, and their mucus-precipitating propensities.

Next to noninsulating property, a vehicle for mucous membrane medication must be of exactly the proper viscosity. Where almost immediate absorbability is desired, as in the case of various dyes and certain other chemical substances which are extremely water soluble, naturally an aqueous vehicle with little or no viscosity may be the most desirable.

However, it is far more common for the physician to use and prescribe combinations in vehicles having a viscosity permitting prolonged therapeutic action, as well as affording some degree of mechanical protection for the mucous membrane. This means that the preparation must adhere to the moist tissues for a sufficiently long period of time to permit absorption of its active ingredients without at the same time interfering with the excessive activity of the mucin-secreting cells during irritated or inflamed states. Neither must the viscosity of a vehicle be such as to permit of excessive spread over the membrane, or its contained active ingredients, molecule for molecule in a given area, will be so much diminished as to markedly adversely influence both the duration and intensity of their action.

Next in importance to noninsulating property and proper viscosity is the solubility of the active ingredients in the vehicle, as well as in the tissue fluids of the mucous membrane and its secretion.

In this connection, Solis-Cohen-Githens[4] point out that the most important factor in connection with the distribution of medicaments in the tissues depends upon their relative solubility. It seems obvious that the word solubility, as used by them, refers to tissue fluid solubility, rather than solution of a drug in a menstruum. Of the pharmaceuticals commonly used for local mucous membrane medication, most are, as a rule, to some degree soluble in aqueous vehicles; some are more soluble in oil, while others are practically insoluble therein.

It is true that by means of various forms of chemical treatment the solubility of some drugs in oil can be enhanced. However, if by means of such procedures mucin-precipitating alterations are produced, then the increased effectiveness of enhanced oil solubility is more than offset by the tendency to produce increased insulation, due to the precipitation of the mucin.

In a general way, if a potent pharmaceutical is soluble in a vehicle at least to the extent of 0.1 per cent, it may produce a definite effect upon the mucous membrane. But, unfortunately, some of our most widely used active ingredients in prescriptions intended for nasal

mucous membrane medication are practically insoluble in oil. For instance, Chen and Schmidt[5] show, and Solis-Cohen-Githens[4] state, that such a commonly used drug as ephedrin is practically insoluble in oils; and it is therefore best used in aqueous mediums.

While it is difficult to appreciate how some drugs which are insoluble in the vehicles in which they are exhibited can render maximal or even adequate therapeutic services when viewed from the standpoint of surface tension and absorptive powers, it is of even greater importance to appreciate that no prescription that is incompatible with the tissue fluids can exert maximal therapeutic effects. Among the foremost requirements for such compatibility is tissue fluid solubility. If the composition of the mucous membrane and the mucus it secretes is approximately 75 per cent water, it is obvious that no oily vehicle will be distributed in these tissue fluids—and no medicament held in an oily vehicle is likely to be able to reach the tissue fluids in an amount greater than is permitted by the spaces existing between the oily globules—and that is apt to be less than 25 per cent of the surface space coming in contact with the mucous membrane.

This is of the utmost practical importance from the therapeutic standpoint when selecting vehicles for localized nasal mucous membrane medication. We must not forget that not only do oily vehicles holding oil insoluble pharmaceuticals tend to physically insulate the active ingredients they may hold in suspension between the oil globules, but also that many drugs depend for their therapeutic action upon electrolytic dissociation within the tissue fluids of their free H or OH ions. In this connection it is common knowledge that oils are practically nonconductors of electric currents, and so far as has been experimentally determined, absolute insulators for the very feeble currents involved in therapeutic ionic intratissue dissociation, even when the electrolyte itself is soluble in the oil. Aqueous vehicles, especially those having a saline content, on the other hand, are good conducting mediums favoring electrolytic dissociation.

This brings us to the matter of hydrogen ion concentration of such prescriptions. Unless the hydrogen ion concentration closely approximates that of the tissues upon which it is to act, a preparation may either become excessively irritating or tend to precipitate the tissue fluids. Moreover, the hydrogen ion concentration, if electrolytes are used, must be such that free H or OH ions are available for dissociation, otherwise preparations containing them are correspondingly decreased in therapeutic efficiency.

The foregoing consideration of some of the factors involved in rational localized mucous membrane medication, while far from being

complete, points out some of the details influencing the pharmacodynamics of prescriptions intended for such use.

Furthermore, it seems evident that while professional tradition and custom apparently plays no little role in the choice of pharmaceutical combinations intended for nasal mucous membrane application, in order to produce maximal possible therapeutic effects it is essential to give attention to scientific pharmacodynamic details. Finally, in view of the many and varied preparations offered the medical profession for this purpose, and the fact that about 47 per cent of physicians' prescriptions, according to Motter[6], are for proprietaries, it might be well to carefully consider the basic principles herein briefly reviewed before accepting or rejecting such preparations as having a scientific basis for rational localized mucous membrane therapy.

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International Digest of Current Otolaryngology.

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Dr. H. C. Rosenberger, Cleveland.

Lange, in the Zeitsch. f. H., N. u. O., 28, 2, 1930, describes a peculiar type of spreading of tuberculosis in the pneumatic cells of the temporal bone. By histological study it was clearly seen that tuberculosis might spread not only through the infection, but also through the exudate in the pneumatic cells. These form tubercles in the soft parts, which may then spread to the walls of the cells.

Kelemen.

Ruttin, in the Zeitsch. f. H., N. u. O., December, 1929, writes on contralateral abducens paresis as an early symptom of otogenous meningitis. He states that abducens paresis on the same side as the otitis is not an extremely unusual condition, but a contralateral paresis occurs very rarely. Ruttin thinks it is due not to meningitic exudate but rather to a toxemia. He describes in detail two cases in which this symptom occurred, both of which developed meningitis and died.

An interesting advance in the handling of medical propaganda was recently made by Congress in Washington when Honorable Anthony J. Griffin, of New York, in the House of Representatives introduced Dr. Arthur McDonald, distinguished anthropologist, of Washington.

Dr. McDonald's work is purely voluntary and disinterested. He is attempting to study a cross-section of American anthropology and its relationship to preventive medicine. He is being assisted by twenty-four specialists from Johns Hopkins Hospital, who have consented to examine gratis every member of Congress who takes the physical anthropological examination of Dr. McDonald. They have

already examined some 100 members and hope to complete the roster in the national capital.

The educational propaganda which these scienists are advancing is based on the plea of corrective medicine and its relationship to anthropology. The character of the subjects as well as the reputation of the examining physicians and scientists speaks for the scientific value that such an examination may bring forth.

Dr. McDonald is interested in having other authoritative groups follow the precedent set by the specialists of Johns Hopkins and will be willing to assist in any way possible. He may be reached at 314 East Capitol street, Washington, D. C.

The Society of Oto-Rhino-Laryngologists, for Lyons, France, and the neighboring district was organized March 1, 1931, with the following officers: President, M. Garel; Vice-Presidents, Prof. Lannois and Dr. Moreau; General Secretary, Dr. Sargnon, 5 Rue Victor Hugo; Assistant Secretary, M. Mounier-Juhn; Treasurer, M. Garnier. Meetings will be held the first Friday of each month in the Salle Pasteur, Hotel Dieu. Membership will include eligible physicians of Lyons and its neighborhood, also corresponding members, both French and foreign.

Waldapfel, in the Zeitsch. f. H., N. u. O., 28, 7, 1930, writes on the subject of the meaning of Fleming's tingiblen Körper (stainable bodies). Unilateral tonsillectomy was done on an anesthetized dog. This tonsil was kept as a control. Then the common carotid and vertebral arteries were ligated on both sides. Twelve hours later the remaining tonsil was removed. Histological examination of the second tonsil showed that the changes produced in the circulatory system had resulted in marked changes in the tonsils. Minimal changes were noted in the epithelium and reticulum, but marked changes occured in the lymphatic structures. There was a marked rarefication of the lymphocytes, together with a tremendous increase in the makrophages which contained untold numbers of minute stainable bodies (tingiblen Körper). Waldapfel interprets the presence of these bodies as having arisen from the cell rests and nuclei, taken up by the makrophages and eventually eliminated by them. KELEMEN.

BOOK REVIEW.

Chronic Nasal Sinusitis and Its Relation to General Medicine. Based on the author's Semon Lecture, University of London, "The Toll of Nasal Focal Sepsis on Body and Mind." By Patrick Watson-Williams, Hon. Consulting Surgeon in Diseases of the Ear, Nose and Throat, Bristol Royal Infirmary; late Lecturer on Oto-Rhino-Laryngology in the University of Bristol; Hon. Member of the Laryngological Society of Vienna; Corresponding Member, Oto-Rhino-Laryngological Society of France, etc. With 109 illustrations. New York: William Wood and Co., 1930. Price $5.00 net.

For many years the medical profession has felt the need of a book written in such a manner that it would show conclusively the intimate relationship of ear, nose and throat diseases to that of the general system, and unfortunately it has been the lot of an ear, nose and throat specialist to supply this need; unfortunately, because naturally his critics will say that he writes with the bias which comes from his ear, nose and throat training.

In reading this book very carefully one cannot help but feel the author's enthusiasm in the meticulous manner in which the subject is handled. Unquestionably he has lived his specialty; practically each and every method that he uses is of his own device. He claims priority for the cytological examination of the nasal sinuses over that of Dean, of St. Louis, and has added his method of "suck-exploration" of the sphenoid which appears perfectly simple; many scientists, however, will approach this technique with awe despite its apparent simplicity.

The chapter on the use of the nasopharyngoscope is most remarkable, being profusely illustrated and bringing out many valuable points not seen in any of our present texts.

The author undoubtedly had the sympathetic help of many internists and general practitioners in those cases of a latent focus of infection in which treatment produced remarkable results.

Watson-Williams describes his own intranasal frontal sinus operation and uses it before resorting to external surgery. When external surgery is necessary he has devised an ingenious operation which, in his hands, obtains the desired results and at the same time is practically scarless.

Generous reference is made to international literature in this work. One omission is noted, however, of a rather important recent advancement, and that is the displacement method of Proetz in the use of lipiodol or of its modification by Fraser.

This unusual book will be read to good advantage by every student and practitioner of medicine.

E. L. M.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

Regular Meeting, Tuesday, Jan. 6, 1931.

Dr. Thomas E. Carmody, of Denver, Colo., was the guest of the evening. He presented an interesting series of slides illustrating X-ray examination of the sinuses in infants and children. These children were under observation for years, during which time their sinuses were X-rayed at frequent intervals. In substance, his remarks were as follows:

Development and Other Studies Upon the Accessory Sinuses of the Nose.

Dr. Thomas E. Carmody.

We know very little about the sinuses. We think we have learned a lot about them in the last ten years, but we have only scratched the surface. A thing that occurred to us a few years ago was that we should begin at the beginning. We had been operating on people 30, 40 and 50 years of age, and a few under that, and we simply saw the last stages of the disease and found the cases ready to die from some other condition. We found the cases when we had a great deal of pus in the nose. We are coming back to a point where we have to do something to make a diagnosis and we have to be very much more careful. X-ray helps us, but we have to use our own power of observation and we have to use many other methods if we want to make a diagnosis that is going to mean anything to the patient.

We heard when Dr. Dean first began doing work on sinuses in children, he said that removal of obstruction in many cases would clear up the condition. In many cases we have seen this true. In other cases, after obstruction is removed, the condition still persists.

In a recent article from Glassboro we have what they call hidden antral disease. This article was brought out by Harper and he speaks a great deal about cases where there is no pus in the nose. I think all of us have seen cases of that kind but I think we do not follow them far enough. In many of these cases we find that the X-ray tells us nothing, or next to nothing. Many of these we refuse to open. Harper has done a radical operation on many of them.

We found in a few cases that in the subepithelial layer we have infection. Even washing many of the sinuses will not tell us much.

Harper has found that in many cases of arthritis which we have all seen, that we have a disease that could be cleared up by operation on the antrum.

Many years ago Shadle, of St. Paul, said that all hay fever was due to antral disease. If he would have said sinus disease, he would have been right. Many of these cases, and I think the great majority, are sinus cases primarily.

About eight or nine years ago Dr. Wassen conceived the idea that many children showed disease early in life, even within the first few hours after birth. He took the trouble to go to one of our hospitals to take these children immediately after birth, some of them one-half hour, some an hour after, and X-rayed their chests. In talking it over with me, I suggested doing the same thing with the sinuses, but not so soon. From that time on we began X-raying the sinuses, some of them two or three weeks after birth, some of them a month, and some not until three or four months after birth. It was surprising how many had diseases in the maxillary sinus. Found one as early as three days. Found one eight days, and very many of them within a year or two or three.

I will now show you some X-ray slides of sinuses in children and tell you something about them.

DISCUSSION.

DR. SKILLERN: I think the pictures just shown by Dr. Carmody are wonderful. He has been able to follow the child's sinuses from infancy through childhood to 15 or 16 years of age.

What are we going to do with these children? Are we going to let them grow up with chronic sinusitis? We can't operate, as this young embryonic tissue does not take kindly to the knife and none of the treatments seem to do any permanent good. I have a little daughter, age 7 years, who has had a maxillary sinusitis for the last two or three years. She goes down to the shore in June or July, and comes back perfectly well in September. A few weeks later she sneezes, usually in the morning at the breakfast table, and out comes a great globule of pus. What are we going to do with such cases? We cannot let these children go on until the entire body becomes a hotbed of infection. If we do they are not going to grow up to be normal individuals.

I would like to ask Dr. Carmody what is his treatment for sinusitis in infancy, then of children between the ages of 5 and 10 years.

I would also like to inquire how he succeeded in getting pictures of such young infants. One of the most remarkable things is the consecutive taking of the same sinus at different ages, thus showing the development of the sinus itself, and the fact that the frontal sinus on the well side develops faster than its diseased fellow, proving that airation has a great influence on the development of the nasal accessory sinuses in man, the same as in the lower animals.

DR. COATES: I have enjoyed and appreciated this talk of Dr. Carmody's and the pictures. There are two or three points that have impressed me: The first is the one Dr. Skillern stressed, that is, the lack of development of the sinuses due to infection. We agree that suppuration in the middle ear in infants does interfere with their development and in the sinuses we would expect the same thing to take place.

Dr. Emerson, of Boston, said he thought it was not the tonsils nor the teeth so much that would cause progressive deafness as the sinuses that did not yield secretion and, furthermore, the sinuses that had atrophic mucous membranes. He found bacteria in the submucosa and in even greater numbers on the periosteum.

As to the connection of allergy with sinusitis, Shadle, many years ago, attributed all cases of hay fever to maxillary sinus infection and "cured" (?) most of his cases by proper drainage of the maxillary sinus. I have always believed that some cases of hay fever and asthma were due primarily to sinus infection. Nevertheless, we must give some weight to what Dr. Proetz said, that in some asthma cases he had taken an X-ray of the maxillary sinus and found that there was no air space and a few hours later found the entire sinus open. It is quite possible that we have, in many of these cases, an allergic condition that is independent of the sinus infection.

DR. RIDPATH: I, with the others, have enjoyed Dr. Carmody's talk very much. I have also been interested in his pictures showing the development of the sinuses in these particular cases.

I think that the history of the infection of the sinuses in childhood, youth and adult stages must be studied more closely than has been done. I feel that the history of sinusitis in people, like the history of many other diseases, can be traced back to their parents, to their grandparents, or back two or three generations. I think we have untoward peculiarities that are not only tuberculous, but there are untoward tendencies and peculiarities of the mucous membrane which is susceptible to infection which can be recognized as a sinus infection in later life. That means a lot of study on our part and also that we are getting down to some basic principle.

What is the cause of that sinus infection? Is it because of the nurse breathing in his face, or has the child some underlying cause? Why is the patient susceptible to this infection, whereas other children can be coughed in their faces and be in close contact with other people having sinus infection and are not infected at all?

Dr. Carmody also spoke of one very important thing that is also important to my mind. I think we are just beginning to scrape the surface of sinus infection. It is only in its infancy. As a matter of fact, it is only in its embryonic stage and I think that food is going to play a very important part, the parents and the environment in which the parents were brought up and the various diseases they had. Vitamins are going to play a very important part, as well as the diseases of the parents and grandparents. I cannot, for

the life of me, see why one individual should be so tremendously susceptible to an infection from an individual and develop within 48 hours a sinus infection, and the other fellow goes through the same thing and does not become infected. There is some reason why one becomes infected and the other one does not.

In Dr. Carmody's pictures, I do not believe all the sinuses are infected. I believe some of these pictures are thickened membranes, that the sinus is filled with a thickened membrane. It may have a mucus discharge but not a pussy discharge. As Dr. Coates stated, this may go down within a few hours. The mucous membrane depletes itself of this discharge and if we take a picture we get a clear sinus. We have to look into these pictures with an eye that is open to conviction, with an eye that is also suspicious.

I would like to hear Dr. Carmody speak on this tendency throughout various generations, while in other families they have no nasal conditions at all. To repeat, this is one of the things we simply have to get down to.

DR. SCHATZ: By the discussion tonight I am reminded of standing on this floor about three years ago when, before this very Society, I read a paper on the "Relation of the Sinuses to the General Economy," and the late Dr. Ross Hall Skillern was the only one that spoke in response to my paper.

In that paper the various factors that should be taken into consideration were mentioned: That the tendency to nasal and sinus disease begins in infancy and lasts practically a lifetime, and that the character of the blood supply is one of the factors, perhaps the most important one.

The teaching of cadaver surgery for years has perhaps led me to think more in terms of anatomy than routine clinical work might have done. One thing noted is the variation in size of the bony foramina through which pass the blood vessels that go to supply the sinuses. Another is the variations that occur in the relative size of vein and artery that supply the same area.

May it not be that such anatomical vascular variations may explain the tendency to pathology in certain individuals? Is it not perhaps the most important factor?

In addition to these anatomical details we must take into consideration the altered physiologic reaction of the blood vessels and the tissue, in certain individuals, to toxic or metabolic agents circulating in the blood. Are not these the underlying factors that explain why some people are sufferers of sinus disease practically all their lives?

DR. STOUT: I would like to ask Dr. Carmody what his results have been where he has operated on the infected sinus, whether the relief is permanent or temporary, whether the relief is almost as good with medical treatment as with surgery.

DR. BURNS: Dr. Ridpath has brought up a question that will possibly be of assistance to me. In the Philadelphia General we have about 400 cases of T. B. and we find that the majority give a history of chronic nasal discharge from infancy. They all tell us they are subject to cold. When you look over a whole series of cases you find they are subject to pronounced nasal discharges. They never seem to go on to become pussy noses. They only advance to a certain stage.

Recently we took a long series of cases and endeavored to find out the family history, and as far as we can interpret these people all seemed to have some predisposition to sinus infection.

In my mind, any time we have cases that become infected so easily, these cases are structurally predisposed to the disease.

DR. CARMODY (in closing): I wish I could be a real encyclopedia so that I could answer all these questions I have been asked this evening.

I don't know whether I should answer Dr. Skillern's question first or not. I might say that I can't answer it in the first place. In my talk, I tried to avoid touching upon the treatment of these children. We try to keep them under the best hygienic conditions—out in the sunshine as much as possible. Many of these children clear up. The treatment outside of the hygienic treatment includes good food, good milk, cod liver oil, and keep the child in the best possible condition. For local treatment we use epanin. I like that personally much better than ephedrin and better than adrenalin. We use a solu-

tion of about one to eight. There are cases where the ostea are too small. Following the above treatment, we have found that the use of solutions of silvol or mercurochrome seem to do some good. It is a question whether they do or not.

Most of the cases are not in the hospital. Most of these child research cases simply come into the hospital and we examine them, take our X-ray and they go home. However, there are cases that we have in the hospital for weeks or months, and in some of these cases, where we find we have pus, we wash them out. You have to be careful not to expose them too much to X-ray. Of course, we have to wash them several times. Sometimes we go to the extent of making a permanent opening. We have done a radical operation on a child age 6 years. These are comparatively rare cases. We always hesitate to do it on account of the embryonic tissue. I do not know whether that answers the question or not. Of course, we find where we have tonsils or adenoids, they should be removed first. We find that many men are removing tonsils and adenoids that do not know how to look into a nose. We find children frequently operated that should have their sinuses washed.

Dr. Skillern also asked how we took these pictures. I do not know. The only thing I know is that we have a Roentgenologist that knows his business. He and his assistants are able to talk to the children. When we get them very early in life there is no trouble. If you get the confidence of the child you will have no trouble. About ten years ago I asked Dr. Allen how he took his pictures. He said he gave the children chloroform. We never do that. No patient of mine has ever been given an anesthetic before being X-rayed.

Dr. Coates said that we have an infection of the sinuses before it reaches the ears. I have often thought that in all this operative work on ears that they should begin at the sinuses, since there are a great many infected sinuses where ears have been infected. We wash out the sinuses and the ears clear up promptly. There are cases that will, in spite of clearing up sinuses, develop mastoiditis.

In regard to the atrophic and hypertrophic conditions, the hypertrophic condition has been found in the membrane. In most of these cases Harper found no pus in any of the sinuses, but infection goes on just the same. I have opened plenty of sinuses that I have punctured and found no pus. The infection is found most frequently in the subepithelial layer.

There are ten infections of the sinuses. The first infection simply interferes with the action of the cilium. The second infection interferes a little with the nutrition of the cilia. In the third infection we have a blocking off of a layer of the epithelium. The tenth infection is when we have ulceration. We get infection clear down to the bone. We can check the stages of progress.

Dr. Coates spoke about Dr. Proetz and the allergic condition. I believe that in most of these allergic cases we have a condition due to infection but also due to having bad grandparents. Somebody has said that in order to be healthy we should choose good grandparents. There is no question that the case I showed here of asthma also showed infection. He has had infection all his life and the infection simply gets worse just before an attack of asthma and he goes on with the asthma attack. It may be that he sleeps on a feather pillow. At any rate, that is what happens.

Another thing Dr. Ridpath suggested was why one patient has this and why another hasn't. Why does one patient die of pneumonia in 24 hours and why does one get well? There is a question in my mind whether or not we all have sinus disease.

Now the point of whether the nurse can carry the infection to the children. I found that in our hospital years ago I tried to keep the nurses that had colds away from the children because their vitality was lowered so much that they could contract it easily. At the present time, in our hospital, the minute the head nurse sees a nurse sniffling, she takes her off duty.

These children that come into the child research council are all examined clinically and by X-ray. We attempt to examine them clinically first and then by X-ray. If the X-ray is made first, I do not look at it until I have made notice of the clinical examination. In 90 per cent to 95 per cent of the cases the clinical report and X-ray report agree.

Dr. Stout asked about the medical treatment of asthma. I do not know about the medical treatment. The cases that come to me are strictly sinus cases. There is nothing to do but surgery. The reason I do not clear them up is because my operation is not thorough enough. I think the cases we do not clear up are those on which we have not done a thorough operation and I do not always do a thorough one.

Dr. Burns spoke about the X-ray not always telling us. We do not know how to read X-rays and I think that if we all had our X-ray man the possibilities are that we would have much better results. In many of the cases that do not show much shadow, when they are opened, we find there is some disease. Harper says especially the atrophic cases do not give any X-ray evidence of disease and in going in he has found much fibrosis.

Now whether the size of the veins or the size of the ostea have anything to do with it, I do not know. In some of the microscopic work that has been done on these people, they found a few cases that had very large blood vessels. In children the sinuses clear up much more rapidly. Grieger, a few years ago, gave us a very profound study of the walls of the maxillary sinus. He showed us about three sets of blood vessels. He showed us that they could be blocked very easily.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

Regular Meeting, Feb. 3, 1931.

Case Report: Hematuria Associated with Acute Mastoiditis. Dr. Ed. K. Mitchell and Dr. M. S. Ersner.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. DUNN: The paper was very well presented and I wish to congratulate Dr. Mitchell, but I think it is very far-fetched. Those of us who have done kidney work know that hematuria will only follow tuberculosis, stab wounds, very severe blows and the acute infectious diseases, such as typhoid, typhus, measles and scarlatina, that also cause an embarrassment of the circulation, but to say that because the cases occur simultaneously with the acute mastoid that the mastoid is the causative factor, is too far-fetched. A child may come in having an acute nephritis and that child may develop mastoiditis or a pulmonary condition but we cannot say definitely that one caused the other.

The mastoid has been blamed for a good many things. If, however, the urine would have been cultured and the very same condition found, we could *probably* trace it to the mastoid.

Hematuria will not last forever if you give rest to the child and give the kidneys the proper water supply. After operation, naturally, the patient has had rest for a week or more, together with the proper diet and after-care, the hematuria will clear up.

I think it is very far-fetched, although the paper was well written and should receive consideration.

DR. SHUSTER: I believe we just cannot pass this over as unimportant because no culture was made of the urine. The fact still remains that after the mastoids were opened the hematuria disappeared. Some of these cases did not have very much fever. Some of the cases of hematuria were on the borderline as far as the mastoid infection was concerned. Something might have been going on that manifested itself in the form of a kidney disturbance. We can use this diagnostic information as another measure to determine whether or not that particular mastoid need be operated.

DR. MITCHELL (in closing): I want to thank the Society for the privilege of presenting this paper. I think the paper will not be in vain if it will stimulate others to look up this subject.

New Test for Borrowed Bone Conduction. Dr. Henry S. Weider.

Dr. Weider reported a new test to differentiate whether bone conduction is direct or borrowed in cases of suspected labyrinthine disease. The Weber test is first performed with localization usually to the sound ear. When the 256 fork is placed over the suspected mastoid and the affected external canal closed, if there is hearing remaining, the sound should become intensified, but if the labyrinthine is dead there will be no effect upon the intensity of the sound. Then the other external canal is closed, producing a marked increase in the intensity of the sound, and often directing the patient's attention to the fact that he is hearing on the well side and not on the side on which the fork is resting. The process is now reversed, the fork being placed over the well mastoid and the external canal on that side closed, causing a great intensification of the sound. After opening the well side, the external canal on the suspected side is closed, with no effect upon the intensity of the sound. This test is particularly valuable in central deafness with involvement of the auditory nucleus or the cochlear portion of the eighth nerve when vestibular apparatus is unaffected.

DISCUSSION.

Dr. Mackenzie: It was my privilege to see this patient with Dr. Weider last week. At that time he told me he intended to present the test referred to at this meeting, but made no mention that he purposed making a demonstration on this patient.

There is no doubt as to the worth of this test. It is new to me. The demonstration made by Dr. Weider is definitely clear. It is a test that promises to be of some value in the future. It recalls to my mind a discussion that took place in Copenhagen the summer before last during the meeting of the International Congress of Otolaryngologists and Rhinologists. Dr. Pohlman attempted to prove that there was no such thing as a lengthening (betterment) of bone conduction in obstructive deafness. At that time I took exception, along with others, to his contention. Among other questions I put to him at that meeting was, how would he explain the lateralization of the Weber test toward the side of unilateral conductive deafness? Also, why when one hearing a fork applied to the skull under ordinary conditions should again hear it after stopping up the ears? The determination of bone conduction is a very important factor in determining the site of the lesion responsible for deafness.

I congratulate Dr. Weider for his conception of the value of the fork tests and his originality in devising the test. At the time I examined this patient, with the noise producer in the hearing ear and the speaking tube in the questionable ear, the patient was unabl to hear the loudest conversational voice. Reversing the procedure, that is, with the noise producer in the deaf ear and the speaking tube in the good ear, the patient was able to repeat the whispered voice, with the mouth of the tube held away at arm's length. Someone suggested that it is possible the patient might be malingering. For this reason I would beg the privilege of Dr. Weider of making the Stenger test.

Dr. Macfarlan: The test presented by Dr. Weider is very creditable. It is a very simple test to remember and I think that if we could conceive that in addition to what Dr. Weider suggested, we could use it as a malingering test, we will have learned more. The malingering test is a very simple test. Your malingerer who says he is partially deaf in one ear will always refer his hearing to the other ear.

To be absolutely sure of total deafness in one ear, the good ear must be damped out completely. The shortening of forks in these tests is that they do not deliver a sufficiently strong tone to be sure that there is absolute loss.

Dr. Weider (in closing): The etiology of the case is what we are searching for. This is unquestionably a toxic case. He has vertigo but the vestibular tract seems to be fairly intact. It may be from his tonsils. We intend to have a differential blood count made. A Wassermann was done with negative results when he was studied at the Polyclinic Hospital.

I think this will prove no malingering case. His answers were absolutely clear-cut. I, having my test in mind, naturally tried him without saying a word of its nature to him. I know he could not have heard about it because nobody knew the test. All his answers were not in the least doubtful.

Paper and Case Presentation: Vincent's Infection of Middle Ear and Mastoid. Radical Operation. Dr. Romeo R. Luongo.

(Appears in full in this issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. BUTLER: This is a very interesting subject. Quite a few years ago I was on the staff of St. Vincent's Home. I do not know whether the name of the home had anything to do with it or not, but we had eight or ten cases of otitis media in which we found Vincent's organisms. We did not cure any of them and I did not have a chance to follow them up.

Dr. Ersner spoke of the severity of Vincent's infection. It may be very mild or very severe.

DR. WEIDER: The fact that there have been so many treatments recommended for Vincent's infection proves that none are universally effective. From my personal experience, I have had better results with a 4 per cent solution of Dakin's oil than with any other treatment. Sometimes I alternate with salvarsan, using one or the other every three or four hours. I have had quite good results.

During the war a soldier had chronic Vincent's. He was in a hospital in England for six months and the condition continued when he came here. He responded very quickly to Dakin's oil.

DR. SHEMELEY: I had the privilege of studying a similar case of Dr. Mackenzie's. We had a child, age 4 years. The case had been diagnosed as diphtheria. The child had had 15,000 units of antitoxin and in spite of this he became rapidly worse. When we saw him he was unconscious. The smear showed Vincent's infection. It seemed almost impossible to operate. One could not see how the child could possibly survive. He was water-starved and had the pallor peculiar to water-starved patients. Dr. Mackenzie agreed to take a chance. The operation was practically without anesthesia. The labyrinth had been destroyed. The child hovered along for several days and finally began to mend. It was about two months after we operated the youngster before we were able to weigh him. At this time he weighed 25 pounds. The child made a subsequent recovery and at this time is residing in West Philadelphia. This case brings one thought to my mind; that many of these cases are misdiagnosed as diphtheria.

Dr. Mackenzie spoke about a discussion in Copenhagen. That entire discussion of Dr. Mackenzie's ,also Prof. Hugo Frey's and Prof. Neumann's, may be found in the recent number of Annals of Otology.

DR. ERSNER: We recently had a case of Vincent's of the sinuses. It started with a fistula posterior to the central incisor leading into the floor of the nose. Endonasal examination revealed a thick exudate on the right side of the septum, which was offensive, and when the exudate was removed a bleeding surface remained. Cultures revealed streptococcus hemolyticus, staphylococcus and pneumococcus. Upon direct smear the Vincent's organism was found. This exudate spread throughout the lateral nasal wall and also involved the opposite side. This continued for about five weeks. In the meantime, we opened the maxillary sinus on the right side and found it filled with foul pus, granulation tissue and generalized bone necrosis. The inferior turbinate was completely sloughed and found loose in the maxillary antrum.

It seems that this type of infection responds fairly well to substances containing oxygen, such as sodium perborate, potassium permanganate and hydrogen peroxid. This patient also temporarily responded to the application of 7 per cent chromic acid solution. These treatments were also supplemented by neoarsphenamin intravenously and local applications of neoarsphenamin.

Dr. Luongo stressed bone destruction. I should like to briefly state that in our case the lateral nasal wall, the nasal bones and all the sinuses were destroyed. Five weeks after the onset the patient developed symptoms of a suppurative central nervous system lesion and while the spinal fluid was pathologic, we were unable to demonstrate Vincent's organisms in it. Two days after onset of the intracranial involvement the patient died.

On postmortem examination we found a perforation through the cribriform plate producing a frontal lobe abscess and secondarily meningitis. The spirochete organisms were found in the brain abscess but not in the meninges.

DR. COLLINS: During the war the cook in the artillery outfit got a primary infection of Vincent's and it spread among the company. All the cases were treated with Dakin's solution as mouth wash and gargle, and all responded to this treatment. This was with Base Hospital, No. 18, A. E. F.

DR. LUONGO (in closing): I have tried peroxid and perborate but the best results are obtained with specific treatments. In the case presented, I could not get rid of the organisms but the symptoms have subsided since I have put her on intense specific treatments.

There are three cases reported that have been cured in 48 hours with applications of sulpharsphenamin. One case is very remarkable. It is a case of acute otitis media where they had positive Vincent's and they say that with one injection of sulpharsphenamin the case cleared up completely.

I want to thank the Society for the opportunity I had of presenting this paper.

Prevention of Acute Surgical Mastoiditis. Dr. Arthur J. Wagers.
(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. STOUT: I agree with Dr. Wagers, but lest someone be led astray, I would like to say that it depends largely on the type of organism we are dealing with. If you have streptococcus or pneumococcus they will go on to mastoiditis, no matter what treatment you give. It is not so much the treatment as the resistance the individual has and the attacking germs.

DR. MACFARLAN: I would like to ask Dr. Wagers if he uses any intranasal treatment in either children or adults, and if he uses any inflation on adults. I would also like to ask what his statistics are on mortality of all classes of acute mastoids. Are there any general statistics on acute mastoids?

DR. SHEMELEY: During the fall of 1918 I happened to be in charge of a camp where we had 1546 cases of middle ear suppuration, complicating flu. It was our practice to attempt to make a diagnosis of acute suppurative otitis media, open the drum and thus provide drainage within the first 24 hours. Of these cases we had only three that developed mastoiditis.

DR. WAGERS (in closing): As to the matter of inflation of the Eustachian tube during an attack of acute otitis media, I have never done it. I have never felt it was the right thing to do. I have feared to force additional infective material into the middle ear. My effort has always been to remove, as far as possible, the infective agents and their products from the middle ear.

Dr. Macfarlan asked about nasal treatment in connection with acute middle ear conditions. This is done routinely, employing some form of antiseptic. As to the rate of mortality in acute mastoiditis, I am unable to give statistics.

THE EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION.

Meeting of Oct. 22, 1930.

Dr. A. F. Holding, Secretary.

At the fifty-first meeting of the Eastern New York Eye, Ear, Nose and Throat Association (composed of specialists of Albany, Schenectady, Amsterdam, Hudson and Rutland, Vt.), held in the city of Albany, N. Y., Oct. 22, 1930, Dr. E. A. Stapleton presented, demonstrated and described his electrical isochromatic dark screen method of taking optical fields. He claims superiority for his method on the following grounds: 1. Only spectroscopically proven true colors are used. 2. The "fixation" and "stimulus" elements are accurately diaphragmed, synchronously lighted, rheostat controlled, spectroscopically true coor lamps. 3. A simple eye-screen distance control. 4. Utilizes the camera lucida for making accurate permanent records quickly. 5. The fields are taken in the dark, eliminating all distractions, such as confusing light reflexes, office paraphernalia, etc. 6. The use of a light in pitch dark has a co-operation compelling influence that a paper disc can never have. 7. Superior accuracy in determining the exact "threshold for colors."

The growing recognition of the importance of field-taking was stressed in the diagnosis of brain tumors; localization of central lesions of the nervous system; the early recognition of multiple sclerosis and the differentiation of eye involvements commonly attributed to sinus infection from more serious central brain lesions.

Dr. Stapleton recommended the works of Lloyd, Peters and Traquair on perimetry.

He summarized the importance of campimetry by quoting: "The ophthalmoscope is the macroscope of the eye, while the campimeter is the microscope of the eye."

The discussion was opened by Dr. F. M. Sulzman and Dr. J. Ivimey Dowling, followed by Dr. Green, Dr. Rainey, Dr. Gabriels, Dr .Marshall and others.

A *viva voce* vote of thanks was extended to Dr. Stapleton for his originality, extended research and demonstration as to the methods, technique and value of field-taking.

An informal round table discussion of the "Detection of Malingering in to the importance of surprise in detecting malingerers, and to the fact that Compensation Cases" was opened by Dr. A. W. Greene. Attention was called simulated deafness is usually unilateral. The reading test, the stethoscope method, the noise apparatus test, the matched tuning fork method (Stenger) and the auditory palpebral reflex methods were all discussed.

THE NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Meeting of Nov. 17, 1930.

Dr. Hilliard Wood, Chairman, Presiding. Dr. H. C. Smith, Secretary.

Malignancies of the Nasopharynx. Report of Two Cases. Dr. J. Leslie Bryan.

Case 1: Mrs. H. T. H., age 63 years, consulted me, giving a history of having a lump in the throat, which was rapidly growing larger, obstructing nasal breathing and causing choking. She had constant headaches and intense pain in the left ear. There was no nasal discharge.

Examination: Eyes normal. Nose: Mucous membrane catarrhal, large septal perforation. Tonsils diseased. Larynx normal. Pharynx showed an oblong mass attached by a broad pedicle to the posterior and left lateral walls, pushing the soft palate forward, extending below the palate, filling the oropharynx. The anterior surface showed extensive ulceration.

Under local anesthesia the tumor was removed with an Eve's tonsil snare. The hospital laboratory reported: "Ulcerated, rapidly growing malignant tumor of the pharynx. Histologically appears to be a fibrosarcoma." A second pathological examination produced a diagnosis of "epithelial carcinoma."

After the removal of the growth nasal breathing was restored and the wound healed rapidly.

One month later the tumor returned in about three-fourths the original size. Irridiation applied to the extent of 400 radium hours caused the mass to disappear entirely in one week's time. One month later the mass had returned, and the patient refused further treatment. Patient died four months after first observation, with diagnosis of acute uremic coma.

Case 2: I. T., age 14 years, gave the history of her nose being stopped up constantly, frequent colds, ears roaring, and nasal discharge.

Examination: Nasal septum deviated to left. Turbinates are hypertrophied, and inflamed with pale margins. Tonsils and adenoids removed with good result. Pharynx showed quite a large mass from the adenoid region extending below the margin of the soft palate, filling the nasopharynx and the upper portion of the oropharynx. Wassermann negative.

A tentative diagnosis of malignancy was made, and a biopsy was performed, with an immediate report of malignancy. Four radium needles of 12½ m.gm. each were applied for ten hours. The next day nasal breathing was fairly well established and the mass in the throat could not be seen without retraction of the soft palate. Within 72 hours the mass had completely disappeared, and nasal breathing was fully restored.

Detailed laboratory report three days later showed the mass to be round-cell sarcoma. Deep X-ray therapy was employed. Four months afterward the nasal passages were slightly obstructed and the patient complained of intense throbbing headaches, which were only relieved by morphin. There was no evidence of a return of the mass in the nasopharynx.

X-ray of the sinuses and skull showed the following: "Both ethmoid regions are cloudier than normal. No clouding of any of the other sinuses made out. The skull shows considerable increased intracranial pressure." While the original tumor has disappeared, there was an extension of a portion that the radium had not reached. Again radium was used, this time a brass capsule containing two needles of 12½ m.gm. each was placed in each side of the nose and allowed to remain 24 hours, giving 1200 radium hours. When the capsules were removed, nasal breathing was re-established, and the headaches had entirely disappeared. The mass in nasal cavities was receding. The condition

of comfort lasted for only a few days, when the old symptoms returned. The patient refused further treatment and grew progressively worse. When last heard of there was complete nasal obstruction, the left eyelids were swollen and closed, and the eyeball protruded.

There was complete loss of vision. The patient had lost weight rapidly and was not expected to live very long.

DISCUSSION.

DR. EUGENE ORR said that he had seen a patient, age 20 years, who could not breathe through his nose. There was considerable growth, which appeared to be malignant. Microscopical examination showed malignancy, and he was treated with radium, which gave temporary relief. He died with metastasis to the liver. During the last year he has seen three malignancies of the nasal cavity. Radium and deep X-ray therapy were used in all of these, giving only temporary relief for several months, after which time the patients died.

DR. HILLIARD WOOD had noted what wonderful temporary relief deep X-ray therapy had given, but that after several months it failed to check the growth.

DR. J. LESLIE BRYAN said in closing that in the case of the young lady who received X-ray in the neck region to prevent metastasis, that it seemed that the metastasis had progressed upward.

Carcinoma of the Larynx; Laryngectomy; Recovery. Dr. Hilliard Wood and Dr. N. S. Shofner.

DR. WOOD: Mrs. H. A. B., age 47 years, first consulted me Feb. 12, 1930, giving a history of attacks of hoarseness for three years, but these attacks had become worse during the past twelve months.

Examination showed the right vocal cord red, slightly swollen and more, or less, granular in appearance. The cord was not paralyzed.

The left cord was slightly red, with what appeared to be an indolent ulcer occupying the middle third of the upper free surface of the cord. There were no other changes in the larynx.

A blood Wassermann and intracutaneous tuberculin test and sputum examination were negative.

Patient was not seen again until Sept. 8. When she returned she was complaining of radiating pain about the right side of the neck, head and ear. Considerable expectoration of mucus, but no blood. No difficulty in breathing and no pain in swallowing. No tenderness, except on the right side of the neck, corresponding to the larynx. Pulse, 88; respiration, 12; temperature, 100°.

Oropharynx and fauces and epiglottis appeared normal. The right side of the larynx, including the right arytenoid and aryepiglottic fold, continuing downward on the right side of larynx, showed a red infiltration. The right vocal cord was not recognizable, except for a white line of ulceration extending along where the free margin of the cord should have been. There was very little motion to the right side of the larynx, either arytenoid or vocal cord; apparently the infiltration interfered with the movement. The enlargement of the arytenoid and aryepiglottic fold had no pallor and no dumb-bell shape appearance—it did not appear to be tuberculous.

The left side of the larynx, including the arytenoid and aryepiglottic fold and left side of the larynx down to near the vocal cord, appeared fairly normal. The left vocal cord had an ulceration along its free margin for the anterior two-thirds of its extent. Only a fairly good view could be had of the larynx and trachea below the true cord, but such a view as could be had showed nothing wrong on the left side below the cord, but some infiltration on the right side of the larynx proper below the right cord.

The right side of the larynx had the most marked pathology, the only noticeable trouble on the left side being ulceration of the left cord.

There was no enlargement about the neck externally, but tenderness when pressed on the right side of the larynx.

Patient was put on mercury and the iodids for three weeks, but was not benefited.

Sept. 26, by means of suspension laryngoscopy, a piece from the right side of the larynx above the vocal cord was removed. This was examined by Dr. J. H. Litterere, who reported the condition to be squamous-cell carcinoma.

Sept. 27, patient turned over to Dr. N. S. Shofner for a laryngestomy. Dr. J. H. Litterere's report after the larynx was removed by Dr. Shofner: "The specimen is that of the larynx, consisting of the thyroid and cricoid cartilages containing the true and false vocal cords and the arytenoid cartilage process. It measures 4 c.m. in length, 4 c.m. in width and 2½ c.m. in thickness. Located at the upper right portion of the entrances is a tumor growth which almost fills the upper portion of the larynx. It fits as a cap over the larynx, and has grown into the vocal cords on the right side, but has left undisturbed those on the left. It is a whitish growth showing some congestion and measures 2½ c.m. in length and 1 c.m in width at the top. It has grown downward into the lateral wall of the larynx on the right side for a distance of 3 c.m. Squamous-cell carcinoma."

DR. SHOFNER (the invited guest of the Academy): Dr. Wood has outlined the history and preoperative findings in this case, including the microscopic diagnosis of carcinoma made by Dr. J. H. Litterere from a specimen removed by suspension laryngoscopy.

The patient was transferred to my service on Sept. 27 for laryngectomy. A two-stage operation was decided upon because of its greater safety. The patient was still uncomfortable and somewhat weakened from the anesthetic and laryngoscopy, and it was considered wise to delay operation for a few days in order to have her in as good condition as possible for operation. The technique recommended by Gordon New was determined upon.

On Sept. 29 the patient was given a duodenal tube, which she swallowed without great difficulty. That afternoon water and food were given through the tube, and nothing except water given by mouth. A special mixture of liquid nourishment was prepared by the dietitian and her caloric intake was kept up to the neighborhood of 2,000 calories per day and the fluid intake to about 3,000 c.c. per day. The patient tolerated the duodenal tube very well.

Operation, first stage: On the morning of Oct. 1 the first stage of the operation was done under local anesthesia. A massive infiltration of novocain was made from the tip of the chin to the suprasternal notch, and a midline incision made in the longitudinal plane. The ribbon muscles of the neck were retracted and the thyroid gland was seen to be greatly enlarged, at least four times the normal size. It was necessary to resect the entire isthmus and a portion of each lateral lobe in order to expose the trachea. The hyoid bone was divided with a Rongeur bone forceps and the two ends retracted so as to expose the upper portion to the thyroid cartilage.

With sharp dissection the larynx and trachea were skeletonized anteriorly and as far posteriorly as was dared, because of fear of injuring the recurrent laryngeal nerves and necessitating immediate tracheotomy. Complete hemostasis was secured and the wound was lightly closed with catgut to muscles and dermal suture to skin, leaving the lower portion of the thyroid cartilage, the cricoid cartilage and first two rings of the trachea exposed. A soft rubber tissue drain was left at the lower angle of the wound.

The patient left the operating room in good condition and the postoperative reaction was not severe, although her temperature did reach a maximum of 101° F. This was improved by codein and steam inhalations of tincture of benzoin. On the fourth day the patient was sitting up in the chair. On Oct. 6, five days after the first operation, in the patient's room the trachea was opened between the cricoid cartilage and the first ring of the trachea and a No. 3 tracheotomy tube was inserted. There was a large amount of mucus coughed up through this tube and respirations were much easier and the patient slept better. There naturally resulted a wound infection, but this was mild and cleared up in about five days.

On Oct. 11 the second stage of the operation was done. A preliminary hypodermic of morphin, gr. ⅙, was given. Anesthesia consisted of a deep cervical block and local infiltration. The original wound was opened and exposed as it

was at the close of the first stage. The trachea was divided completely at the site of the tracheotomy and the cricoid cartilage and the trachea and larynx were dissected out from below upward with scissors. The thyrohyoid membrane was divided transversely with a knife and the specimen removed. The upper end of the esophagus, where it had been separated from the larynx, was sutured to the free edge of the thyrohyoid membrane with continuous plain catgut, thus closing the pharynx. The divided trachea was sutured to the skin with interrupted silk, and the skin brought together in the midline with interrupted mattress sutures of silk. A soft rubber tissue drain was placed below the tracheotomy and the tracheotomy tube left in trachea. A large gauze roll was placed on either side of neck and tightly strapped in place with adhesive tape.

The anesthesia had not been as complete as one could wish, but in spite of the patient having experienced some pain, there was no shock and the patient left the operating room in good condition.

There was a rather marked temperature reaction, the peak being 102.6° the second day, but it quickly subsided to 100° and gradually returned to normal. There was a rather severe tracheitis and bronchitis, but no signs of pneumonia. There was a mild wound infection, which rather quickly subsided. There was a salivary fistula, which had its exit just above the tracheotomy and necessitated constant packing, but this gradually closed and on Oct. 26 was completely closed. Oct. 28, 17 days after operation, water and fruit juices were taken by mouth, and on Oct. 30, 19 days postoperative, the duodenal tube was removed and the patient has been on regular diet since, without any difficulty with deglutition.

On Oct. 15, four days postoperative, patient was sitting up in chair, and a few days later walking.

From time to time the tracheotomy tube was left out, with the hope that it could be left out permanently, but there was always a slight retraction of the edges of the opening, and the patient became nervous and preferred having the tube replaced. She larned to care for the tube herself and even removes the outer tube and reinserts it with the aid of an obturator.

On Nov. 12, 31 days after the second operation, the patient was discharged in good condition, free from pain and very happy. She has learned to speak in a fairly audible whisper.

DISCUSSION.

DR. W. G. KENNON said that this was probably the first laryngectomy ever done in Nashville. Biotomy was desirable before such a mutilating operation as a laryngectomy was done. He had read of a similar case, where all of the history, symptoms, etc., pointed to malignancy, and where a laryngectomy had been done, only to find the condition was tuberculous. It was wiser to do a biotomy and then do a laryngectomy if it was necessary.

DR. EUGENE ORR said that the most striking feature of this case was the bilateral involvement of the vocal cords.

DR. HILLIARD WOOD said that his report tonight was merely a transcript of his record and did not include the laboratory report in detail. In Dr. Litterere's report he said that the trouble was confined to the right vocal cord, but with the mirror Dr. Wood had thought that he saw an ulceration. He had had three cases of malignancy of the larynx in a year, this one being the only one who lived.

THE NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

Meeting of Feb. 16, 1931.

Dr. Hilliard Wood, Chairman, Presiding.

Report of Bronchoscopic and Esophagoscopic Work for the Year 1930.
Dr. Hilliard Wood and Dr. W. W. Wilkerson, Jr.

Dr. Wood and Dr. Wilkerson reported eleven cases in this series; ten of the patients gave a definite history of having swallowed a foreign body and in none the foreign body was found and removed. Of the two cases in which no foreign body was found, one patient was a neurotic, probably hysterical.

Of the nine cases in which the foreign body was found and removed, the youngest patient was 8 months old; the others were 2, 3, 4, 8, 9, 64; two were 45 years of age. There were seven males and four females; nine white and two colored patients.

The time the foreign bodies were retained varied from three hours to five weeks, the average time for the nine foreign bodies being eight days. In six of the cases the foreign body was retained 24 hours, or less.

The foreign bodies were located as follows: Six in the esophagus, one in the larynx, one in the trachea, and one in the left bronchus.

The foreign bodies found and removed were: four bones, two open safety pins, and one each of the following: metal disc, shoe button and chestnut hull.

The anesthetic used was ether in eight cases, and local anesthesia in one. Of the eleven patients, ten recovered with slight or no complications. There was one fatality. This child was 2 years old and was supposed to have swallowed a grain of coffee eight days before. Marked bronchial symptoms and paroxysmal breathing had developed. The X-ray findings were negative. An exploratory bronchoscopy was done with a 4 m.m. bronchoscope. No foreign body was found, but a well developed bronchitis was present. Ten hours later the patient had grown rapidly worse, the most marked symptom being difficulty in breathing. A tracheotomy was done under novocain anesthesia and for a time the condition improved, but 24 hours later, that is, about 36 hours after the bronchoscopy, the patient died, with the symptoms of bronchopneumonia.

In addition to the above, there were five cases of stricture of the esophagus and one of cardiospasm. Four of these had been treated from time to time for two or thee years; only two were seen for the first time during 1930. Two small children have been treated for three years with retrograde bouginage for lye strictures of the esophagus. The strictures were dilated up to a 30 F. size, which is regarded as ample, as the children are only 5 and 7 years old, respectively.

Three lye strictures of the esophagus occurring in white men of middle age were all caused by concentrated lye taken with suicidal intent. Two of these were seen for the first time in 1930, and are being treated with retrograde bouginage. The advantages of retrograde bouginage in the treatment of lye strictures are that there is no danger of perforation, and the treatment is less disagreeable to the patient than dilatation from above.

THE MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

SECTION OF OTOLARYNGOLOGY.

Meeting of Jan. 9, 1931.

Dr. John Brown, President, presiding.

Sarcoma of the Larynx. Dr. J. A. Watson.

Dr. Watson reported two cases of sarcoma of the larynx.

DISCUSSION.

DR. W. E. PATTERSON stated that he had been much interested in these two cases reported by Dr. Watson, particularly the first case, as he had had frequent opportunities of seeing the case and occasionally giving treatment.

During the past few days, after having attempted to locate some current literature by a thorough search of textbooks and periodicals of the Hennepin County Medical Library, he was surprised to find evidence of an almost complete dearth of informative literature on this subject and but scattered case reports in the old literature, which led him to believe that sarcoma of the larynx is much rarer than is generally supposed, or indicates a marked laxity on the part of clinicians to report their cases. He said that with but one exception the most recent case report found was in the *British Medical Journal*, of 1915. (This statement refers to English publications.) In the A. M. A. Quarterly Index two references were found of case reports in German periodicals which were not available in this library.

In the 1928 Year Book, M. Slobodnik is credited with having reported a case operated in 1926 in the Virchow Hospital. A laryngectomy was done and the patient was reported to be in good health six months afterward. Fauvel found seven cases of sarcoma among 300 cases of laryngeal growths. Gurlt reported one case of laryngeal sarcoma among 848 cases of morbid growth in the body. Bosworth reported having found 47 cases recorded in the literature, but admits that in his extensive practice he had but one case.

Other statisticians, Hahn, Krause and Schmidt, give the incidence of sarcoma of the larynx to be about one to 25 of carcinoma. The histological data gives the most common form to be the spindle-cell variety, while fibroidangio- adeno- and lymphosarcoma are infrequent.

In the *Medical News* dated Aug. 29, 1884, Louis Jurist, of Philadelphia, writes of having seen a laryngeal growth of grayish tint and dense consistency about the size of a horse chestnut springing from the left ventricular band, which proved to be a sarcoma. This patient died from suffocation before surgery could be instituted two months after the onset of symptoms.

In the Reference Handbook of Medical Sciences, published in 1887, Dr. Patterson said he found a fairly extensive treatise by Butlin on the subject of sarcoma of the larynx.

In the *Medical News*, of 1895, John A. Thompson, of Cincinnati, reports a case on which he operated twice. This patient died of hemorrhage due to lung metastasis two months later.

Halstad, in 1896, reports a sarcoma originating from the right aryepiglottic fold, the size of a walnut, in a man age 56 years.

About the same time Bliss, of Philadelphia, describes a large fungoid growth which proved to be a small round-celled sarcoma on the epiglottis of a man age 55 years. This patient lived one year after tracheotomy was done.

Dr. Morgan McWhinnie, of Seattle, in 1910, reported the case of a man age 54 years, where the tumor was in the form of two pedunculated masses, one below and one above the cords near the anterior commissure.

Dr. Cowie, of Bournemouth, England, reports an interesting case in a man age 75 years with a sarcoma involving the right side of the larynx, extending downward from the right aryepiglottic fold. He was operated by the thyrotomy method, the growth being carefully dissected away from the thyroid cartilage. Six months later the patient apparently was free from any recurrence.

Data was very incomplete as regards final results of treatment, as the longest period of freedom from recurrence reported in any case operated was six months. It appears that this disease is almost surely fatal, but, unless the growth is in such a location as to obstruct breathing or swallowing, the patient may live for a considerable length of time because of the fact that ulceration is not usually extensive and there is little tendency to metastasize.

As sarcoma occurs with greater frequency in early life, it was surprising that all specific case reports found in the literature were of men past middle age. As Nature seems to mask the sarcoma cells by superimposing apparently benign fungoid or granulomatous growths, it is extremely important to get repeated biopsies in all cases where the least suspicion exists, and it is very evident that nothing short of a radical and thorough extirpation of the growth and all surrounding tissue involved promises the least prospects of a cure.

DR. G. C. DITTMAN asked if the glands were involved in the first case.

DR. WATSON stated that never at any time were the glands involved.

DR. W. E. CAMP asked as to the duration of time between the first biopsy and those sent to Dr. Ewing.

DR. WATSON replied that the first specimens were taken by another clinic about six months before he had seen the patient, and Dr. Watson had seen him at various times for about one-and-a-half years before these specimens were sent to Dr. Ewing.

DR. CAMP said he was under the impression, as Dr. Watson read the history, that some time had elapsed between these reports, and asked Dr. Watson if he did not think it possible that in that time sarcoma could have been superimposed upon this other tissue.

DR. WATSON stated that the fungi found so persistently were possibly the cause of the original growth, which seemed to consist for so long of granulomatous tissues purely. Dr. Bell's reports were always the same and undoubtedly the sarcomatous degeneration was a secondary process. In spite of the first pathologist having insisted that it was carcinoma, Dr. Ewing's report on specimens taken both early and late always agreed with Dr. Bell's.

Dr. Watson called attention to the fact that citromyces are not supposed to be pathologic in man, but were found frequently, he believed, in horses and other lower animals. This man was occupied a great deal around horses. Possibly that was the source of the trouble in the first place; the irritation and inflammation from the presence of the organisms developing eventually into sarcoma.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. D. Harold Walker, 5 Bay State Rd., Boston.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.
Place: Briarcliff Lodge, Briarcliff Manor, Westchester County, New York. June 18-20, 1931.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. Francis R. Packard, 304 S. 19th St., Philadelphia.
Secretary: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Place: Atlantic City, N. J., June 15-17, 1931.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY.

President: Dr. Max A. Goldstein, 3858 Westminster Pl., St. Louis.
Secretary: Dr. R. L. Loughran, 33 E. 63rd St., New York.
Place: St. Louis, June 3-4-5, 1931.

AMERICAN BRONCHOSCOPIC SOCIETY.

President: Dr. Henry B. Orton, 24 Commerce St., Newark, N. J.
Secretary-Treasurer: Dr. Louis H. Clerf, 128 S. 10th St., Philadelphia.
Place: St. Louis, June 6, 1931.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

President: Dr. William H. Wilder, 122 S. Michigan Ave., Chicago.
President-Elect: Dr. J. F. Barnhill, Miami Beach, Fla.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: French Lick, Indiana, Sept. 14-19, 1931.